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TIPPING REACTION IN CASES OF VERTIGO AFTER HEAD INJURY

AN OTO-NEUROLOGICAL STUDY

By

KARL RÅDMARK

Stockholm

1944

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AN OTO-NEUROLOGICAL STUDY

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PREFACE.

My interest in tipping table tests — as a part of clinical oto-neurological examination — was aroused in 1934 by Professor C. O. Nylén, then assistant chief of the Ear, Nose and Throat Out Patient Clinic of the Serafimerlasarett (Head: Prof. G. Holmgren), and it was on his initiative that a number of patients from the Neurological and Neurosurgical Departments were examined. The cases were mainly ones of nervous diseases and of cerebral tumor or the like. The first results were reported at the annual meeting of the Ear, Nose and Throat section of the Swedish Medical Association in 1938 (Published in Nordisk Medicin 1939).

In 1939 I started to work on a series of vertigo cases after head injury. During the last three years the work has mainly been conducted at the Ear Department of the Sabbatsberg Hospital, Stockholm. It has taken a long time, partly because I tried to follow up the patients with later examinations and partly because I wanted to await the result of corresponding examinations on a large number of cases suffering from producer-gas poisoning in order to check the accuracy and objectivity of my observations.

Professor C. O. Nylén has been of invaluable assistance to me with his great knowledge and experience in otoneurology. As his pupil in this sphere I wish to offer him my warmest thanks for all his willing help with good advice and factual criticism.

I owe a large debt of gratitude to Professor N. Antoni for his friendliness in placing patients at my disposal, for helping me with literature and for his good advice in neurological matters.

The examination table was placed at my disposal thanks to the help of Professor Torsten Skoog, and I also wish to thank him warmly for helping me with his good advice.

My chief, G. Öhngren M. D., has tried to facilitate my work in every way. Time and apparatus have been placed at my disposal thanks to him and he has aided me with good and practical advice. For all this I thank him most warmly.

I wish to thank Dr. RAGNAR BRINGEL for the patients he has allowed me to examine and for all his willing assistance in trying to register the different tipping reactions. At the same time I wish to thank my colleagues at the Ear Department, Dr. L.-E. FLOBERG and Dr. STURE HJORTH, for all their self-sacrificing help in the examinations. Dr. L.-E. FLOBERG provided his camera and took all the films, for which I owe him a debt of gratitude.

I wish to offer my warmest thanks to the foundation "Stiftelsen Therese och Johan Anderssons Minne". It defrayed the cost of the examination table and contributed to the cost of filming the tipping reactions.

Stockholm, December, 1943.

Karl Rådmark.

Introduction.

Thanks to Rademaker's and Garcin's investigations and writings in 1932 and 1933 entitled "L'Épreuve d'adaptation statique" we now possess a method of examining the central vestibular connections with the help of a tipping table which supplements and enriches the usual method of carrying out otoneurological examinations. Hitherto the form of induced vestibular reaction given the most attention has been nystagmus. Examination on a tipping table gives us a method of observing more objectively the extremity reactions in the arms to caloric, rotatory and other forms of irritation.

A number of uncertain factors in tipping table reactions — particularly their connection with and relation to the other vestibular reactions in the otoneurological status — have made it an uncertain and unreliable method of clinical examination. The present work has been compiled in order to some extent to clarify these factors. It only covers, however, a small sphere of otoneurology viz., examination of patients suffering from vertigo after head injuries. — By vertigo is meant a disorder in the patient's relationship to his surroundings in space in a statokinetic respect which does not involve a loss of consciousness.

Otoneurological tests are still very imperfect, being both uncertain and subjective. Various writers emphasise in different ways, however, the importance and value of the pathological signs and symptoms, although the methods vary tremendously with the different investigators. The publication of the present work therefore has a small ulterior motive, as it is designed to contribute towards a standardisation, as far as that is possible, of otoneurological testing for vertigo after head injuries. With this object in view, more space has been devoted to the methods employed and to the discoveries arrived at than would have been

necessary had the work only sought to set forth the results of examinations on the tipping table.

The records of the cases reported in the table on page 74, on which the discoveries announced in this work are based, have been deposited in quadruplicate in the Upsala university library where they are at the disposal of interested readers. They have not been translated into English.

CHAPTER 1.

A Review and a Critical Analysis of Earlier Literature.

a. Experiments with Animals.

Goltz (39) and Ewald (33) showed a long time ago that a pigeon, the semicircular canals of which had been destroyed on both sides, was not capable of the muscle reactions necessary to maintain its balance if placed on a stand which was rapidly shaken or moved. More recently, André-Thomas (93, 94) among others has experimented with dogs. During the first few weeks after the section of the eighth nerve on both sides, dogs displayed no equilibrium reactions either to slow or rapid movements of the examination table. The animals were placed on all fours on a table which could be tipped through an axis in the horizontal plane both longitudinally and transversally to the direction of their bodies. Some time after the operation, the animals reacted to slow table movements. This was attributed to impulses of non-labyrinthine origin. Their inability to react in a normal manner to rapid movements of the table remained unchanged, however.

Rademaker and Garcin (72), Tait and McNally (91, 92) and de Kleyn and Versteegh (50) have studied equilibrium reactions rather on the principles which underlie the use of tipping reactions as a method of testing the labyrinthine functions. The former showed, like André-Thomas, that after bilateral labyrinthectomy dogs lacked the automatic muscle reactions necessary to preserve their balance when the table on which they were standing was shifted rapidly, whereas their reactions to slow movements were normal. This examination involves rotation through the fronto-occipital and bitemporal axes in the course of which the vestibular-induced extremity reactions are studied. Rademaker showed that after unilateral labyrinthectomy there was no reaction to movements to either side for several days,

but some time later the animal regained its power to react normally as before the operation, and a state of complete compensation developed. Animals deprived solely of their cerebellum manifested clear extremity reactions to both slow and rapid movements of the table. The experiments as a whole showed among other things that the labyrinth is a special organ for the maintenance of balance during rapid changes of position and that the proprioceptive and extroceptive reactions serve the same purpose during slow movements. Similarly performed balancing tests with animals deprived of their cerebellum have shown that the main function of the cerebellum is in this case to regulate and coordinate the muscle reactions arising from irritations of the labyrinth so that they become well balanced and effective movements for the preservation of balance. Despite advanced ataxia, an animal deprived of its cerebellum displays clear defensive reactions. After labyrinthectomy the defensive reactions disappear and the cerebellar ataxia becomes more pronounced. Labyrinthectomy does not appear to cause a reduction of the static muscle tonus but on the contrary a reduction of the kinetic in such a way that each labyrinth affects the supporting tonus on both sides of the body. This fact may perhaps explain why compensation can occur after unilateral destruction of the labyrinth if the labyrinth on the other side is normally functioning. The muscle reactions are then produced on the right side of the right posterior and left anterior vertical semicircular canals and on the left side of the corresponding left posterior and right anterior canals.

John Tait and McNally (91, 92) have conducted admirable investigations in this sphere on frogs, isolating various parts of the labyrinth and then carrying out tests on a special tipping table. Following the principle which was mentioned above in connection with Rademaker's examination of the functions of the vertical semicircular canals during tipping, the animal was placed diagonally on the table in order to obtain a differentiated irritation of the labyrinth. It is safe to say that the rôle of the vertical semicircular canals in tipping reactions has been clearly demonstrated in the case of frogs by these really admirable operative and experimental examinations.

The experiments on rabbits and guinea pigs conducted by de Kleyn and Versteegh (50) gave on the whole the same result i. e. that it is the vertical semicircular canals which probably play the principal role in the inducement of extremity reactions during

rapid tipping. The short period of latency also suggests that reflexes of the semicircular canals are involved. TAIT expresses his view as follows (91, p. 286): "This then is the first great characteristic of semicircular function, namely, that appropriate responses are made — quick, rapid, almost lightning-like — so as immediately to counteract sudden tilting and jerkings of whatsoever kind. The canals are called into action by something sudden; their effect is momentary and evanescent."

b. Clinical Examinations.

On the basis of experiments on animals, RADEMAKER and GARCIN have carried out tipping tests both on normal persons and on pathological cases. During these tests the patient was placed on hands and knees on a stretcher which could be lifted at the sides. The various normal cases reacted more or less in the same way to rapid tipping to both sides, with stereotyped movements which were the same on both sides. Tipping through a transverse axis also yielded very small discrepancies of no practical significance. A special tipping table was used by them as long ago as 1929, the movement lying in the plane of the table top. This can be seen in an essay dated 1933 (73) where this method of investigation received the name "L'Épreuve d'adaptation statique".

The different writers place their patients in the same position during the tests, but ZADOR (101, 102) has also conducted tests on patients in other positions: sitting, standing, prone and supine. Since the reactions thus obtained do not correspond in any way with the reactions which can be experimentally induced in the vestibular region and which serve as a check on the objectivity of the tipping test, this method of examination is of no major importance. I will substantiate this assertion at a later stage. Following the method adopted with animals, TAIT and MCNALLY placed the patient diagonally on the table. This is doubtless right in theory, but it is not certain that it is feasible to apply to human material methods which are physiologically correct with animals. Nor was the differentiated reaction which might have been expected obtained in practice.

The tipping movements have been executed both rapidly and slowly. In the former case the effect on the semicircular canals is displayed in extremity reactions which, with small variations, are more or less the same in all healthy people. The slow move-

ments are primarily designed as a check and in order to explain the test to the patient. All investigators have adopted the method of giving the table a rapid initial movement followed by a gradual slowing down after a certain angle, but ZADOR (102) constructed a special device on his table which permitted a gradual acceleration followed by a sudden stop. This method does not give the impression of having been very suitable, however. A gradual acceleration of the tipping rather induces a chain of reaction where one chain of reflexes overlaps the next until the extremity reaction is complete. First come the extroceptive and proprioceptive impulses, followed perhaps by the functions of the utriculus and finally of the semicircular canals, if the threshold value for the latter is reached. The sudden braking of the movement against a hard object is apt to result in abrupt and jerky reactions on the part of the patient, obliterating all nuances in the manner of reaction. We have not found experiments with this method to yield any advantage.

As far as methods are concerned, DE KLEYN and VERSTEEGH (50) used a table in their experiments which was of a very different type from that used by the others. They placed rockers under the table, which partly changed the principle of the table movements. With normal patients, however, the tests yielded more or less the same results as those conducted by other writers.

The reactions of normal patients to rapid tipping have been practically the same in all cases. RADEMAKER (71) discerned three phases in the reaction, and these are more apparent in pathological cases, particularly where there are disturbances of equilibrium due to central injuries with a ceberellar-vestibular genesis:

1. The "labyrinthine phase", being the sudden muscle reaction due to the irritation of the semicircular canals induced by rotation.

2. The "passive phase" due to the shift of centre of gravity. The body is moved passively to one side.

3. The "proprio-extroceptive phase" is the final one and is due to the body's attempt to achieve stability in its new position. The deep and cutaneous sensibilities then assert themselves. In-coordination, assymetry, asynergia, etc. and other atactic phenomena occur in certain conditions. This phase of the reaction is very valuable from a diagnostic point of view.

TAIT and MCNALLY (91) examined deafmutes and in two cases found an absence of post-reaction to the Barany rotation as well

as a pathological tipping reaction. No differentiated reaction with loss of reaction for a certain semicircular canal or a plane of semicircular canals was specified.

RADEMAKER and GARCIN have published two works, the first in 1932 (72) and the second in 1933 (73). The first gave details of the results of experiments with 22 cases and the second of about 80 cases. The patients suffered principally from nervous diseases such as tabes and cerebellar disorders. In all these cases the patients displayed normal tipping reactions. Patients suffering from cerebellar-labyrinthine disorders, either unilateral or bilateral, displayed pathological tipping reactions in every case, and at the same time a pathological reaction to ordinary vestibular stimulation of either the horisontal or vertical semicircular canals. A number of patients suffering from so-called "labyrinthine disorders" were also examined and displayed pathological tipping reactions. The patients were mainly suffering from multiple sclerosis, but there was also one recent head injury. The vestibular status was not either fully normal in these cases. A couple of cases of compensated unilateral labyrinthine disorders were examined and the tipping reactions were normal. The labyrinth was dead on one side but the other functioned normally. They have seen some patients with spontaneous vestibular signs such as nystagmus, defective gait, past pointing and vertigo who reacted pathologically on the table without induced irritation yielding any pathological signs from the eighth nerve and its connections. They point out in this regard that the tipping test seems to them to provide a more sensitive indicator of vestibular injury than other methods of examination. The latter cases were ones of unilateral, non-compensated vestibular injury. They also mention a case of "encephalopathie infantile" which yielded a pathological tipping reaction but which at the same time displayed no clinical indication of a labyrinthine vestibular disorder. A closer examination showed, however, pathological deviation of the arms, past-pointing, falling tendency and defective gait. They point out that such conditions are interesting since they may show that the results of different tests to induce extremity reactions coincide.

The material in the book which RADEMAKER (71) published in 1935 is much the same and provided no directly fresh ideas on the subject of tipping reactions, but he says in conclusion that the examination can be made equally well in bed or on a stretcher and that no special table is required. He also considers that the main

purpose of this method of examination is to ascertain whether or not the ability to react exists. How the patient reacts is not so important.

Zador (102) has in his work studied the development of tipping reactions during the years of childhood and considers that the position on hands and knees is not physiologically correct in the case of adults. Rademaker does not believe this, and it is not really so very important if the position is not physiologically correct. The main thing is that the organ which is irritated — the labyrinth and its connections — should be subjected to physiological influence. This cannot in his opinion be denied.

De Kleyn and Versteegh (50) conducted their tests on a table of a different construction and examined both normal and pathological cases. They examined a couple of cases of head injury, one of which was not fully compensated until 4 years after the accident, but in the second case compensation came as soon as 5 weeks after the injury. — They also mention a couple of cases where there was no extremity reaction to ordinary vestibular tests and the tipping reaction was also pathological. They consider that a case of congenital syphilis which displayed no reaction to caloric and rotatory irritation but a fully normal tipping reaction shows that these phenomena do not always coincide. For safety's sake this case was also examined on a Rademaker-Garcin table but the tipping reaction was still perfectly normal.

Zador's investigations apply mainly to nervous cases which were often very complicated and therefore difficult to judge on the basis of his descriptions, but he also examined a number of deafmutes whose induced pointing ability was nil. When examined on a tipping table, they reacted normally in the sitting and standing positions but in some cases pathologically when on hands and knees. The deafmute genesis is not known for certain. He claims to find no connection between the clinical vestibular discoveries and the pathological tipping reaction, even in the pointing experiment, but declares that "la déviation des index n'est pas un indicateur valuable du manque d'adaptation" (102, p. 127). In his view the absence of any reaction to tipping tests appearing at the same time as normal nystagmus reactions and the fact that the contrary can also be proved in the ordinary vestibular and tipping tests also indicates that there is no direct correspondence between different vestibular functions.

Rademaker and Garcin maintain that in cases of a pure cerebellar syndrome the tipping reactions are normal, but Zador maintains that this is not always the case but that there is both hypermetria and dysmetria, as is typical for such diseases. This can also be demonstrated in cases where there is no other definite indication of injury to the cerebellum with the result that examination on a tipping table is of great importance for the purpose of diagnosis. He shows furthermore that the reactions in cases of cerebellar disorders closely resemble those of children in their second to third year where incomplete cerebellar functioning physiologically gives the same reaction picture.

Zador's material largely resembles that of Rademaker and Garcin but he also examined some cases of extrapyramidal syndrome combined and not combined with cerebellar symptoms, cases of stupor and idiots without somatic neurological symptoms. The first-named yielded infantile reactions and a difference in the reactions of head, body and extremities, presumably connected with hyperkinesia. Stupor cases reacted normally. The last-named yield infantile reactions but their equilibrium is relatively in advance of their undeveloped mental state.

Zador sums up his impressions at the end of his great monography, with particular regard to the localising of the injury where the tipping reaction is pathological, as follows:

a. Destruction of the labyrinth, injury to the eighth nerve or its radicular fibers and to vestibular nuclei or connections yield pathological tipping reactions to rapid tipping; if the injuries are bilateral and total then the absence of reaction is permanent and total. Are they partial, however, some form of reaction occurs in one way or another; after compensation the reaction is normal.

b. "Our knowledge of the effects of injury centrally above the eighth nerve is incomplete. We know that injury to the posterior columns of the spinal cord (tabes) does not cause a pathological tipping reaction. Theoretically it may be assumed that injury to the centrifugal vestibular pathways to the spinal muscle centres for extremity movements might cause a pathological reaction. We know very little about these connections, however, nor do we know of any case where a spinal lesion affecting these supposed connections has caused a pathological tipping reaction without at the same time causing muscle paresis."

As a neurologist pure and simple, however, Zador has not been able to arrive at the true state of affairs. The material has probably

not been analysed sufficiently rationally from an otoneurological point of view.

c. An injury situated centrally above the vestibular centres in the pons causes secondary modifications of the equilibrium reactions. This may be present in cases of cerebellar symptoms, extrapyramidal hyper- or akinetic syndromes etc., and so is rather a topographic modification based upon symptoms. In cases of cerebellar changes, one sometimes gets more pronounced secondary symptoms on one side if the injury is more pronounced on that side.

Finally, he points out that the tipping reaction does not only depend upon the labyrinthine function but also upon the centres of motor coordination. It is the collaboration of various nervous components which gives satisfactory results. This is best seen in the study of secondary modifications for, according to ZADOR, they involve no vestibular disorder. However, he regards the reactions from a purely neurological point of view with the result that the vestibular factor has been forced into the background.

A number of other investigators and clinical workers have described examinations and tests on tipping tables, but rather in the form of a recapitulation of the above works without any original views. STENVERS (85) has mentioned them, WEISZ (97) has examined children and in this country STURE BERGGREN has for many years examined his vertigo cases according to DE KLEYN and VERSTEEGH. The latest work in which we have found the tipping reaction mentioned as a method of examination is by VAN DEN BOORN (22) but it contains nothing new about the method.

c. Summing up and a Critical Survey.

Experiments on animals have revealed among other things that:

1. After double labyrinthectomy there is no defence reaction to rapid tipping movements through a fronto-occipital (craniobasal) and bitemporal axis. The failure to react is permanent. ANDRÉ-THOMAS (93, 94, 95), TAIT-MCNALLY (91), RADEMAKER-GARCIN (71, 72, 73), DE KLEYN-VERSTEEGH (50).

2. Unilateral labyrinthine destruction causes absence of reaction in the same way as before during the first period after the operation, after which there is an improvement in that the animals for a time react normally to one side. After a further period com-

pensation occurs so that there is fully normal reaction in all directions.

3. The reaction involved is a reaction originating in the semicircular canals, presumably the vertical semicircular canals. The utriculus and the sacculus have nothing to do with the reaction. TAIT-MCNALLY (91, 92), DE KLEYN-VERSTEEGH (50) and RADEMAKER-GARCIN (69, 70, 72).

Clinical use of a tipping table for examinations has yielded the following, *inter alia:*

1. that in examining normal material a reaction is obtained which is typical, uniform, but with a reservation for movements occasioned by the special stato-kinetics in which human beings differ from animals. According to ZADOR (102) the manner of reacting in the hands-knees position corresponds phylogenetically more closely with that of monkeys;

2. that in examining pathological cases with defects analogous to those mentioned in groups 1 and 2 above, corresponding reactions are obtained, within certain limits, in experiments on animals;

3. that central vestibular changes also occasion pathological tipping reactions;

4. that it has not been possible to prove any certain correlation between vestibular changes and tipping reactions except in cases of peripheral labyrinthine injury. RADEMAKER and GARCIN (73, 71) and DE KLEYN and VERSTEEGH (50) suggested the existence of a correlation in cases of central injury;

5. that it has not been possible to prove any connection between the position of the injury and the direction of the defective tipping reaction;

6. that according to RADEMAKER and GARCIN (73) the reaction progresses through several phases and that cerebellar-vestibular injuries sometimes occasion specific reactions. Idem, ZADOR (102);

7. that examination on a tipping table is a valuable vestibular test since it in some cases yields a reaction which indicates vestibular injury without its being possible to prove the existence of such injury by any other method. RADEMAKER and GARCIN (73,71).

RADEMAKER and GARCIN have proved that pathological tipping reactions can be manifested by cases which otherwise display ordinary vestibular reactions at the same time. One such case was of "syndrome labyrinthique pur", probably post-encephalitic, which displayed pathological tipping reactions but no "anomalie

appréciable" in reacting to ordinary classical vestibular tests (Observ. V. Tex. 72, p. 10.). The case in question was one of bilateral central vestibular disorder, the genesis of which is doubtful, however. But they also observed similar reactions in cases of unilateral central vestibular affection in which cases the patient had vertiginous crises as though afflicted with tumour, multiple sclerosis or the like, and was examined on the table during the intervals of recovery. The normal classical tests have not indicated any "anomalie évidente" with respect to the eighth nerve. The signs of the disease were nystagmus, defective gait, past pointing and attacks of vertigo (73, p. 571). No casuistic data exist and so it is difficult to form any idea of the nature of the disorder. They mention two similar cases in their work published in 1935 (71, p. 118), one of multiple sclerosis with "hyperexcitabilité vestibulaire diffuse" and the other of "syndrome labyrinthique" without any specified cause and with "hypoexcitabilité du labyrinthe gauche", but neither display any defence reflexes to rapid movements on the tipping table. They conclude from these cases: "Les réactions des extrémités peuvent donc être absentes alors que les réactions des épreuves classiques sont présentes."

They consider that the tipping reaction is in some cases more sensitive than other vestibular reactions but they have not proved that there is any correspondence between the various vestibular reactions. They mention two cases, however, where it is possible to discern a correlation between dissociated vestibular signs and a pathological tipping reaction. In one there was fracture of the skull with deafness on the right side and defective hearing of unknown genesis on the left together with "réactions assymétriques" to a rotation test (71, p. 114) though these reactions are not described in detail, and another was a beautiful case of "encephalopathie infantile" with cerebellar symptoms which gave a pathological tipping reaction but which under ordinary induced vestibular examination gave normal nystagmus but no pointing reaction, vertigo or defective gait. They point out also that this case is worthy of notice because it showed corresponding vestibular reactions in the matter of extremity reactions to the various forms of vestibular stimulation. De Kleyn and Versteegh also have similar cases with complete absence of extremity reaction both under ordinary vestibular tests and when examined on the tipping table. They mention one case, however, which reacted in the opposite manner to the above-mentioned Rademaker-Garcin

case in that it yielded a fully normal tipping reaction but no vestibular response to caloric and rotatory stimulation. This case has been mentioned previously. It concerns a 29 year old patient with congenital syphilis (50, p. 48). — According to these various results the tipping table reaction does not directly correspond with other vestibular reactions, and ZADOR points out this fact after his own examinations. According to DE KLEYN and VERSTEEGH (50, p. 51) it is safe to claim that the tipping reaction occupies a place of its own in relation to other vestibular reflexes because:

1. "RADEMAKER and GARCIN have shown that the tipping reaction may be pathological while the ordinary vestibular tests do not show any pronounced vestibular defects.

2. DE KLEYN and VERSTEEGH mention one case in which ordinary vestibular examination revealed absolutely no labyrinthine function on either side but which yielded a normal tipping reaction."

How can these two observations be explained? The tipping reaction is a defence movement originating from the vestibular area. It issues from both the periphery and the centre as a result of stimulation of the semicircular canals or their central areas and pathways and it must therefore be correlated to a reaction occasioned by other means in these areas. RADEMAKER and GARCIN have pursued this line of thought and they state, with reference to the reactions to ordinary vestibular examination in the above-mentioned case, that it was not possible to prove an "anomalie appréciable" or "évidente". This would suggest that the results of ordinary vestibular tests are treated with some reserve. Furthermore the material has probably not been analysed in such a way that corresponding reactions could have been obtained i.e. there has been no differentiated vestibular test. The observations made in the case with "encephalopathie infantile" show that they were on the look-out for the true connection between extremity reactions occasioned by different forms of vestibular irritation. DE KLEYN and VERSTEEGH had a couple of similar cases and thought that the absence of extremity reactions to classical vestibular irritation and to tipping tests corresponded. They could not say anything definite however, and they are not convinced of the truth of their claim. Thus with regard to RADEMAKER's and GARCIN's cases, the answer to the question beginning this paragraph is that the cases have not been sufficiently carefully examined from an oto-neurological point of view, i.e., they have not been examined

for dissociated vestibular reactions in the matter of pathological extremity responses. — De Kleyn's and Versteegh's case is on the other hand more difficult to explain. In this case there is congenital syphilis and this sometimes causes a partial absence of vestibular function while other vestibular functions in the same organ at the same time remain normal. Rauch (75), Ruttin (77, 78), Kleestadt (46). I have seen quite recently a couple of cases of syphilis with normal hearing, normal caloric and tipping reactions and a total absence of all rotatory postreactions. Since it is known that syphilis can cause such discrepancies between various vestibular reflexes this may explain the facts in this case. The eight deafmutes whom Zador examined manifested similar discrepancies and Zador points out in connection with their examination that he could find no correspondence between the vestibular reactions and the manner of reacting on the tipping table. Presumably, however, these cases can to some extent be cited as proof of this correspondence and as further proof that examination in the hands and knees position — which he does not consider physiological in the case of human beings — is the only one which proves anything from the point of view of the vestibular functions since it provides a check-up on the reaction by giving a corresponding extremity reaction to classical vestibular irritation. If there exists a syphilitic genesis, however, the result must be treated with reserve owing to the consequent possibility of a partial failure of the vestibular functions.

The above remarks apply only to reactions to rapid movements of the table since reactions to slow movements are of no importance as extremity reactions from a vestibular point of view.

Zador has by his work widened the knowledge gained by Rademaker and Garcin regarding the nature of tipping reactions in clinical examinations of cerebellar complications in vestibular disorders. This is very valuable because these conditions are very usual. Dysmetria and asynergia are often manifested in defence reactions without the signs of ataxia in ordinary testing being particularly prominent. It is possible to differentiate between the various phases in the reaction. Firstly there is the vestibular reflex from the semicircular canals and thereafter an adjustment and adaptation reaction which is secondary to the changed static position. Zador (102, p. 121) has discerned two further phases in this latter phase, firstly a passive movement due to the displacement of the body, and secondly a proprio-extroceptive phase

where the patient tries to correct the new static position with the cutaneous and deep sensibilities as a partial driving force. It is possible that the utriculus also has a certain function in these stabilising reactions. The main feature of the phase, however, is the assymetry and the poor coordination which is very apparent in a one-sided cerebellar affection. This can be seen in both rapid and slow table movements but not so clearly in the latter.

CHAPTER 2.

My Own Investigations.

A. The Object of the Investigation and the Working Hypothesis on which it is Based.

In vestibular examinations one seeks first to ascertain in which ear i.e. periphery apparatus the defect that may exist is situated and then to arrive at a differential diagnosis on the side where the defect has been found, that is to say one seeks to arrive at a differential diagnosis of the labyrinthine or rather vestibular affection in question, since the defect may be a purely central one. We have hitherto had BÁRÁNY's method with caloric and rotatory stimulation to examine the functioning of the semicircular canals (ampullae) and by combining these methods one can sometimes locate an isolated defect in them. Actually one can differentiate between two different reactions in the semicircular canals, one of which has nothing to do with gravitation in its widest sense, for instance rotation through a vertical axis which gives rise to reactions from the horisontal semicircular canals, and the other which provides protection against gravity in the form of extremity responses, the movement being in this case through a horisontal axis. In cases of defective balance, it is possible for the purpose of differential diagnosis to distinguish between these forms of reactions in the semicircular canals. In the first case, rotation through a vertical axis is obtained by using an ordinary swivelling chair and in the second case the rotation through a horisontal axis by examination on a tipping table. The former yields reactions from the horisontal semicircular canals and the latter from the vertical, with the result that these two examinations complement each other (TAIT, 91. p. 292). Ordinary caloric stimulation provides separate responses in the same ear from the horisontal semicircular canal on the one hand and from both the vertical semicircular canals together on the other, and rotation in a certain manner yields a response from

the plane of the horisontal — isolated from response from the vertical — semicircular canals. The latter provokes a response from the semicircular canals on both sides, the horisontal being responsible for one plane, the posterior vertical on one side and the anterior vertical on the other for the other plane. In these examinations the principal reaction takes the form of a movement of the eyes — nystagmus on examination in various positions of the head. The reason for this is the direction of movement of the lymph in the semicircular canal in relation to the ampulla. Thanks to NYLÉN (64, 65), DE KLEYN and VERSTEEGH (49) and others we know that various positions of the head can of themselves cause nystagmus, so that the result of an examination of this kind can sometimes be complicated and not fully satisfactory. The result has been that in order to complement this classical method of vestibular examination, efforts have been made to exploit the reactions occasioned in other muscles than those of the eyes, i.e. in the extremities, to see if another method might yield results which could be used to complement or check these discoveries. If it were possible to check up on the deviation and the pointing reaction in the arms which arise from caloric and rotatory stimulation in what is for normal cases a definite manner by the use of a special and more objective method of examination, then still more profit could be derived from the ordinary vestibular tests. One method of examination would then serve as a check on the other. The irritation of the sense organ concerned — the semicircular canals — must be identical i.e. a flow of lymph must be set up, but the irritation must be effected according to a different method. By observing more closely the nature of the extremity movements resulting from different forms of vestibular stimulation one might obtain a better method of judging the vestibular functions both in normal and pathological cases. The observation of nystagmus in vestibular tests is of course extremely valuable, but the significance of the state of the extremity reactions which it has hitherto not been possible to judge with such great certainty owing to various difficulties will in future acquire greater importance because we have obtained in the tipping table a method of examination with which to verify their objectivity. It supplements in a happy manner the results obtained in ordinary classical vestibular tests but can in some cases provide information about the condition of the eighth nerve and its connections which we could scarcely obtain without its help. A rapid tipping movement

gives rise to extremity reactions which proceed through different phases and in some pathological conditions we obtain characteristic changes in *these* phases which are of diagnostic importance.

Emphasis has been laid above upon extremity reactions as a particular object of examination, and the tipping table has been brought into use to provide a check on them by means of another form of vestibular irritation. TAIT's experiments on animals show that with the aid of this examination one can arrive at a differential diagnosis of a labyrinthine disorder because the different vertical semicircular canals manifest characteristic reflexes where there is isolated injury. This applies to his experiments with frogs but one cannot arrive at such a differential diagnosis when it is a case of vestibular conditions in a human being. The correspondence with animal experiments lies in the total and permanent loss of defensive reactions to rapid movements of the table after bilateral loss of the functions of the vertical semicircular canals and the same temporary condition after a corresponding unilateral loss, on condition that the semicircular canals on the other side are intact. RADEMAKER and GARCIN and DE KLEYN and VERSTEEGH have reached this result. Otherwise complications arise as soon as this method of examination is applied to human material. The result is that one asks oneself:

When is the tipping reaction pathological?

Why is the tipping reaction pathological?

Is there any correspondence between the reaction obtained in examination on a tipping table and that which is obtained in an ordinary classical vestibular test?

The above investigators have already attempted to answer these questions, and, as I pointed out when summing up, certain cases have indicated correspondence between the various reactions. RADEMAKER and GARCIN have one case in which a pathological tipping reaction appears at the same time as absence of deviation of the arms, past pointing and defective gait after caloric and rotatory stimulation. DE KLEYN and VERSTEEGH (50, p. 48) have a couple of similar cases regarding which they write as follows: "Bei diesen Fällen macht es den Eindruck, dass parallell mit dem Fehlen der reaktiven Erscheinungen an den Extremitäten bei den verschiedenen vestibulären Reizmethoden auch die Extremitätenreaktionen beim Kippen nicht auslösbar sind." Despite his thorough investigations ZADOR has not been able to find any correspondence between the results of his various vestibular

examinations. He places particular emphasis upon this point and in fact claims that (102, p. 127) "la déviation des index n'est pas un indicateur valuable de manque d'adaptation". This statement refers to his description of the result of an examination of deaf-mutes in all of whom he found a loss of pointing reaction and in some a pathological tipping reaction when examined in the hands-knees position. He obtained reactions which corresponded in some cases but not in others and this is partially analogous to DE KLEYN's case with congenital luetic bilateral loss of the labyrinthine functions. Nor did this case yield any correspondence between the reactions to ordinary vestibular stimulation and to examination on a tipping table. It seems not improbable that the reason for the discrepancy between the manners of reacting in ZADOR's case is the same as that which is highly probably in DE KLEYN's and VERSTEEGH's, viz. syphilis. The reaction on the tipping table of a peripheral vestibular injury is one with which we are well acquainted as a result of the foregoing investigations, but it is not absolutely clear from the above and from other sources what is the effect of a central vestibular disorder. The object of this work has therefore been to try to obtain, after vestibular tests and examinations on a tipping table of a large number of cases displaying vestibular symptoms arising from central injury, reactions which correspond and which might be used to answer the above questions. In addition it should give some guidance in certain other matters connected with the value and usefulness in otoneurology of this special method of examination.

During the last ten years prominent research workers and writers have devoted much work to tipping table examinations. Different pathological cases with or without vestibular symptoms have been systematically examined on the table — as was previously mentioned in chapter 1 — and one might think that after such extensive and thorough investigations as those conducted by RADEMAKER and GARCIN and by ZADOR there would be nothing more to add. That is not the case, however, for they, as physiologists and neurologists, have not penetrated the tipping reaction from an otoneurological point of view. The classical vestibular test has usually fallen into the background. Information about this is scanty and any studies of the differentiated vestibular reaction as may have been carried out have usually not been complete. The object seems mainly to have been to ascertain whether or not there existed any vestibular function and the

principal interest has been attached to the nystagmus reaction. In any case the information regarding the vestibular status is in most cases accompanied by such reservations as "appréciable, évidente, hyper-, hypoirritabilité etc." The above is not written in order to criticise, but on the contrary only in an attempt to explain why the different vestibular reactions have not been found to correspond.

B. My Cases.

The various writers cited above have largely made use of similar cases and this has perhaps contributed to the results obtained. As a rule they have made use of nervous cases of a more or less complicated nature, but in view of the method of examination adopted by, for instance, ZADOR, who only sought to ascertain the appearance of the tipping reaction in different pathological conditions, this is of course fully understandable. I began myself in 1934 at the ear out-patient department at the Serafimer Lasarett working upon similar hospital cases from the neurological and neurosurgical departments, but it was usually impossible to gain any idea of why the patient reacted in such and such a manner (RÅDMARK, 79). The condition was frequently not constant and the results of checks made at certain intervals varied. Frequently, furthermore, the condition of the patient was such that this check could not be made. In cases of multiple sclerosis one sometimes found that the tipping reaction corresponded with deviation and past pointing brought about in the usual way, but it was often difficult to judge the whole thing with any degree of certainty. The condition was variable and unsteady. The nerve and tumour cases which were otherwise examined gave no result beyond what was already known thanks to the writings of RADEMAKER and GARCIN. Various kinds of vertigo cases of nervous origin were examined both during the crises and during the calm intervals and yielded approximately the same result as the examination of the multiple sclerosis cases. The diagnosis and the genesis were frequently unclear and the patients were often impossible to examine *lege artis*. The result of all this was that I tried to obtain material for examination which was as free as possible from disturbing neurogenic complications and as constant as possible and in which the disorders were more or less due to the same causes. Cases of dizziness after head trauma proved suitable from these points of view and there were plenty of cases. They also had

one advantage, their tendency to a certain limited degree of healing and restitution which could make it possible to investigate the internal relations of the vestibular reactions in this respect.

The material on which this work is based, therefore, consists of cases of dizziness after head injury where the trauma itself is often of ancient date and where dizziness in one form or another is the dominant symptom which remains. Every case has been carefully examined neurologically either by a practising neurologist or at the neurological departments of the Serafimerlasarett. As a rule they have been cases of accidents during work referred by the insurance companies to neurologists for an examination and a medico-legal opinion. After making his own examination, the neurologist has referred the case for oto-neurological analysis with an enquiry as to whether the dizziness is somatic or psychogenic, that is to say, whether it is possible to prove by otoneurological methods the existence of organic injury in the eighth nerve and its connections which might conceivably be due to the trauma in question and which might also be the cause of the dizziness. This has led to an otoneurological examination of the cases which has included an obligatory tipping table test.

The material embraces 140 patients, in 60 of which the existence of organic vestibular injury has been proved. In two cases it has not been possible to prove any vestibular injury but the symptoms pointed to intracranial organic disorder of "vascular" genesis without any pathological condition in the vestibular region. Seven cases when examined on the tipping table reacted as cases of vertigo due to psychical causes usually react and it has not been possible to find any organic vestibular defect in the status otherwise. The remainder — 71 cases — proved fully normal under vestibular examination, and their tipping reactions have also been normal. The table below displays the composition of the material:

Organic vestibular disorders	60
" intracranial disorders without pathological vestibular reactions	2
Reactions due to psychical causes	7
Normal reactions	71
Total number of cases examined	140

The material is almost exclusively male, which is presumably explained by the fact that the injuries were due to accidents.

Table 1.

Age distribution years	16—20	21—25	26—30	31—35	36—40	41—45	46—50	51—55	56—60	61—65	66—70
Number of cases . .	2	1	7	9	12	10	8	9	6	4	1

The ages of the patients vary between 19 years and 67 years. The age distribution in the cases with normal reactions is about the same as that in three other groups combined. It is these three groups which are of interest in this work.

The table shows the distribution of ages in this combined group. The largest number lie between the ages of 36 and 45 years. Only 3 are under 26 years. 38 are above 40 years. This is presumably connected with the fact that head injuries do not display the same tendency to cause vertigo in youth, at any rate not at so late a stage as in older age groups. This is supported by SKOOG's (84) investigations which involved the examination at a later date of children with skull fractures, all of whom displayed normal vestibular conditions.

It may also be a coincidence that my material has come to be composed in this way, and no great importance should therefore be attributed to it. The neurologist has sifted the material sent in for examination and grave injuries, i.e., cases with clear organic symptoms and injuries confirmed for instance by X-ray, have frequently not been submitted because an examination has not been considered necessary for judging the case.

The nature of the external injury is of minor interest in this work as also the question of whether or not the existence of a fracture has been proved in accordance with SKOOG's (83) examinations. In most cases there is probably a fracture even if it is only confirmed in a few cases. A definite commotio cerebri is common to them all. I do not attribute such great importance to these matters in the disease history but attach all the more weight to the symptomatology with particular reference to vertigo. All suffer from vertigo in one form or another, either spontaneous or arising from certain movements or position. As a rule the patients are well able to describe their symptoms, particularly where the vertigo is due to certain movements or positions. Frequently the descriptions are more or less coloured by a desire on the part of the patients to further their own interests in relation to the insurance

company with the result that their evidence must be treated with great reserve. Particular complaints can often, however, be verified by oto-neurological examination.

One cannot clearly distinguish the vertigo types from one another, for the boundary between one type and another is fluid and one must almost regard vertigo as a sign — a symptom — of different degrees of severity in a comparatively similar injury, Sture Berggren (20). It is of course all the time a question of injury to the same functioning mechanism, namely the vestibular apparatus. The past history provides no definite indication of the vestibular location of the injury, but one obtains information as to the form of vertigo; spontaneous or induced, and its manner of expression: a diffuse feeling of uncertainty, a feeling of movement (ordinary rotation) of the subject or object and the direction of this movement in space, its connection with other symptoms such as buzzing, defective hearing, faulty sight, nausea, vomiting, etc., which are of a certain value. The past history should be able to provide preliminary information about the vertigo which can usually be confirmed or disproved during the examination which follows. One can divide it into two groups, one originating from the peripheral areas and one from the central. The latter can again be divided into two, one of which is typical for a central vestibular injury involving changes in or in the vicinity of the vestibular nuclear areas and affecting the vestibular tracts and pathways in different directions. A form of vertigo is reported in the anamnesis which is comparatively typical and which has been called "vertigo of the brain stem" by Stier, Beyer (21, 87—90). The other manifests a form of vertigo which is more complicated since it is dominated by symptoms which are not of a purely vestibular nature but must rather be regarded as a sign of "general vascular influence on the brain". In this group, rotatory vertigo often does not appear until sometime near the end of the chain of symptoms which afflict the patient and then frequently in such a mild form, or rather so overshadowed, that many do not at once notice it. The most pronounced symptom in this group of vertigo of central origin is the feeling, particularly after certain movements or in certain positions of the head or body, that things are going black in front of the eyes — the patient feels "misty" and has to rest. If the trouble progresses, vertigo of a rotatory nature accompanied by nausea and vomiting often develop. Since the patients often recognise the first symptoms in a disorder of this kind and so

learn to protect themselves against the final phase, one does not hear this latter spoken of so frequently. — The above has been included because it is of importance in some respects to the oto-neurological examination which is to follow.

We know the way in which a patient with a peripheral vestibular injury reacts on the tipping table, and no such case has been included. It is also a fact that accidents seldom result in isolated peripheral injury for the blow, however slight it seems to be, causes at the same time a modification in the brain. Consequently, the cases of peripheral vestibular disorders are in this work combined with central disorders. There are six such cases. The combination of symptoms which can be described as of central origin and dependent upon organic lesions affecting the vestibular centres or tracts and pathways in various directions is of greater interest. It is also the most usual in practice and in the present series, in which sixty cases have been collected together under the name "brain stem vertigo" or "brain stem injury".

It is comparatively rare that in head injury material one comes across the vertigo which is provoked by "general vascular brain injury" for it is rather seldom of traumatic origin even if a head injury may have contributed to its appearance. Its origin should actually be sought in changed nutrition and blood circulation in the brain on a large scale, which changes it is impossible at least oto-neurologically to locate. Hypertonia and arteriosclerosis are doubtless the most usual causes. The vestibular status is normal in this case, and the group should actually not belong here but has been included because vertigo occurs and particularly because the tipping reaction bears out that which this work attempts to prove. The material includes two such cases.

One might think that the material should symptomatologically be fairly uniform and simple in a vestibular respect when one can divide up the vertigo into groups, but this is not the case, for the grouping is only preliminary and embraces a great variety of symptoms and reactions. — I have found it suitable to study the tipping reaction in these cases merely because vestibular disorders predominate in contrast to the material which I examined before. In a case for instance of multiple sclerosis, tumour, etc., vestibular symptoms can appear with the same predominance as in head injury material, but more sporadically and often complicated by symptoms of another central disorder of a rapidly chang-

ing nature, making it difficult and uncertain to compare the tipping reaction with other vestibular reactions. Vestibular troubles after a head trauma often appear at an advanced stage of the illness when the patient is beginning to get up or trying to return to work (MYGIND 63). They are stationary for a long time, displaying the same symptoms and reactions, and the modification in the subjective and objective condition usually occurs rather slowly and systematically. There are of course exceptions but they are doubtless in most cases complicated by a nervous, non-organic disorder which it is the task of the internist and neurologist to discover for himself. (DE CRINIS, 31.)

The time which elapsed between the injury and the examination varied between one month and nine years, but most cases have come between a year and a year and a half after the accident. No recent cases have been included, although some have been examined. This is because it has in many cases not been possible to conduct the examination in the manner which is desirable if reliable, measurable results are to be obtained. For humanitarian reasons, one cannot drive a patient with a skull fracture, commotio cerebri, out of his bed for an oto-neurological examination which is often both unpleasant and strenuous for a person in his condition. Nor is there in most cases any medical symptom which calls for an examination of this kind at so early a stage so I have refrained from carrying it out except in certain special cases. — Actually, it is of subordinate interest for the purpose of this work when the patient is examined in relation to the time of the trauma, for the most important thing is merely to be able with patients suffering from organic vestibular injury to demonstrate a correlation between the pathological reactions obtained by ordinary classical vestibular tests and the reactions to a tipping table examination. The age of the patient might perhaps have some effect upon the tipping table reaction, but the normal material shows that this is not the case.

The symptomatology of the case, from a vestibular point of view, and the fact that it has been possible to exclude by neurological examination internal disorders other than those arising from trauma, with the result that the material has become as "pure" as is possible from the point of view of the vestibular function, were the essential and fundamental considerations underlying the choice of this material as a suitable working object. The symptomatology will be mentioned more closely later on.

Table 2.

Approximate interval between injury and examination	1 month	1 ½ month	3 months	4 months	5 months	6 months	7 months	10 months	1 year	1 ½ year	2 years	2 ½ years	3 years	4 ½ years	5 years	6 years	7 ½ years	8 years	9 years
Number of cases . .	4	1	2	7	2	6	2	2	8	6	12	1	7	2	1	1	1	2	1

For the sake of completeness a table is appended which shows the number of cases examined and the intervals which elapsed between the time of the accident and the time of the examination. The time is given in months and years and comprises an approximate, round period for each case. The table covers the cases included in the combined group.

C. Method of Examination.

Since there has been a neurological examination of every case in this material, special steps have been taken to try to find an organic explanation of the trouble in the cases where such action seemed grounded. Encephalograms were taken in a number of cases and the blood and cerebrospinal fluid were examined. Serologic testing for syphilis was done in all the cases, but they have all yielded negative results. The neurological examination also included psychological tests as well as an ordinary internal medical examination. This primary examination of the patients was of great value since it excluded a number of pure nerve cases from examination and has resulted in the genesis of the disorders being uniform. This does not of course prevent the symptoms from sometimes resembling those of an organic nerve disease of one kind or another, but the neurological analysis could not establish organic origin. In cases where the oto-neurological examination yielded results pointing in this direction, the patient has been returned for a fresh examination, but the result has usually been the same.

Koch (55), among others, considers that a constitutional nervous factor plays an important rôle in the appearance of certain vertigo symptoms after trauma with characteristic reactions to vestibular tests. This applies to the vestibular tonus difference of central origin where the changes in the objective and subjective

symptoms are great and varying and where it is possible that a nervous disorder of some kind may underlie the manner of reaction. This fact is mentioned merely to point out that there may perhaps exist certain nervous conditions which are of importance in the appearance of certain post-traumatic vestibular subjective and objective symptoms the existence of which cannot be proved by neurological examination. KOCH'S assertion is merely a hypothesis but it is very possible that it is correct. One may hope that an improved method of examination will be able to verify it and to explain both the manner of reacting and the symptoms.

The normal cases used in this work were neurologically and medically examined in the same way as the pathological cases. The series comprises 52 cases, all examined on the tipping table. All the patients reacted normally, and their neurological and otoneurological status was also normal.

The vestibular examination has been thorough and strenuous both for the patient and the examiner. As a rule, it has not been carried out in one sitting but has been spread over a couple of days, after which a check has in many cases been made a couple of days later. The examination has been designed to discover both spontaneous and vestibular signs and reactions due to special stimulation. The hearing tests have been conducted in the "silent room" at Sabbatsberg hospital, both in the ordinary way with tuning forks, whispering and conversation, and with an audiometer in the cases where the hearing defect was uncertain.

1. Spontaneous Signs.

Ataxia in the arms, past pointing and deviation of the arms, an abnormal Romberg sign in various forms, different kinds of gait defects, pathological corneal reflexes and spontaneous nystagmus have been counted as spontaneous symptoms. In all cases different tests have been conducted to discover these signs, and they reappear in the neurological report, with the result that the examination on this point serves in part as a check. The corneal reflexes proved to be of no importance for the purposes of this work; the whole material does not contain one positive case. The other signs are more or less subjective, with the exception of nystagmus, and they cannot either be ascribed any diagnostic importance except in connection with purely objective reactions.

The ataxia tests were included, however, in order to see whether the results tallied with the manner of reacting on the tipping table. The same is true of the pointing tests and the tests for static and kinetic equilibrium disorders. The methods employed in these examinations were the usual ones. Special interest was devoted to the pointing test because it was thought that its results might correspond more closely with the manner of reacting during a tipping table examination.

The patient's tendency to exaggerate is often extremely large in cases of this kind and one must be most careful during the spontaneous tests not to class as pathological anything which does not give the same result when the reaction is induced. One does not therefore derive any great immediate benefit from the examination for spontaneous reactions. The only purely objective spontaneous sign which we have in this case is nystagmus, and that has in every case been examined under BARTHEL's spectacles in the usual way.

The pointing test was carried out according to the GÜTTICH-WODAK method in such a manner that the patient was placed on a chair without arms, the patient's head held facing straight ahead. Arm movements must be loose and easy, and clothing etc. which causes obstruction is therefore removed. Displacements of the centre of gravity must be avoided and the patient must sit in a straight and symmetrical position. He is told to point straight ahead with both arms parallel and then to move his arms slowly up and down, returning each time to the original position on a level with his shoulders. This is first done a few times with open eyes, after which the patient does the same exercise with his eyes closed. Deviation and pointing error are measured in centimetres, the former being decided first, after which the arm movements are begun. The patient, therefore, must not be influenced manually to point in any given direction, and the examiner should not stand in front of him and give the orders. This manner of examination was used all through the tests, even in induced pointing. Other pointing tests were also carried out, but we found the one just described to be most rational. Before the test one should explain to the patient what he is to do and assure oneself that he has understood. His deviation and pointing error should not be corrected, but the test be repeated with and without the eyes' help.

2. Induced Vestibular Signs and Reactions.

These have been examined by:

a. Examination for nystagmus and vertigo in various positions and after changes of position.

b. Caloric and rotatory stimulation.

c. Optomotor stimulation.

d. Examination on a tipping table.

a. Examination for Nystagmus and Vertigo in Various Positions and After Changes of Position.

All cases were examined for nystagmus and positional vertigo and the tipping table proved suitable for this purpose, since it lessens the risk of cervical reflexes and circulatory disturbances. The patient lay still upon the table, which was tilted to different sides. The nystagmus obtained was classified according to NYLÉN's (65, 66) type grouping, which largely corresponds with that of FRENZEL (37). Type I is a nystagmus which beats in all directions, usually changing direction when the head is moved in the opposite position. Type II is marked by a nystagmus in a definite direction, influenced by and often tied to a certain position. "Transitory nystagmus" was not observed in the present series. Rotatory or vertical nystagmus in the head hanging position only, without any other demonstrable alterations in the vestibular condition occurred. The nystagmus is then often accompanied by dizziness and in some cases this condition was considered typical for the vertigo group "general vascular brain injury". It was included because the tipping reaction like the rest of the vestibular reactions was normal, but the only objective sign of a central defect was this positional nystagmus, positional vertigo. Nystagmus is of subordinate interest to tipping tests but it has been included partly in order to complete the symptom picture for purposes of diagnosis and partly because nystagmus tests belong to the routine examination. Nystagmus — both spontaneous and positional — is of course a purely objective reaction which indicates a peripheral or central organic disorder, and can in certain circumstances be of the greatest importance to a vestibular diagnosis. NYLÉN and BERLIN (68) drew attention to the importance of positional nystagmus, describing one case in which it was the only objective oto-neurolog-

ical sign. — It may be pointed out in passing that one sometimes discovers a latent nystagmus by taking the patient's head in one's hands and rotating it rapidly to right and left and then bending it back. This procedure was mentioned by BRUNNER (25—27) as "Kopfschütteln" and in America has received the name "head movement test". It has proved useful, particularly in post-traumatic conditions, for revealing a latent nystagmus (GROVE, 41, MURPHEY, 62).

b. Caloric and Rotatory Stimulation.

A series of methods of examination, including bilateral irrigation according to RUTTIN, were tried out, but the following procedure appeared to be the most rational and was therefore used in all the cases.

After otoscopic examination the patient was placed in a chair with his head inclined backwards at 60 degrees. The auditory canal was syringed with 10 cc. of water at 27 C. for ten seconds, the tip of the syringe being passed along the full length of the posterior upper wall of the auditory canal. The anatomic conditions of the patient's ear were ascertained before the examination. The irrigation was carried out as identically on both sides as is possible. BARTHEL's or FRENZEL's spectacles were used in the nystagmus examination and the patient looked straight ahead or a few degrees to one side in the direction of the expected nystagmus, the procedure being the same for both sides. After irrigation, the time elapsing before the appearance of the slow nystagmus phase (period of latency) was recorded and the direction, form, amplitude, frequency and eye deviation, if any, were observed and noted. The length of the period of latency and the duration of the nystagmus were recorded. — — — Then the patient was allowed to rest for at least five minutes before examinations on the other ear. If the examiner was uncertain about his observations or thought that he obtained a pathological reaction, the examination was repeated after a suitable period of time and in exactly the same way.

Examination for deviation, pointing error and falling was carried out during the same sitting or else after another similar irrigation. The examiner looked first for deviation and then for faulty pointing, during which slow arm movements were executed as stated previously. Otherwise the writer followed BEHRMAN's (18) and

WODAK's (98, 99) method, which is a modification of BÁRÁNY's (1, 11, 12).

If a clear vestibular reaction (nystagmus, deviation, past-pointing, falling tendency, vertigo) was not obtained when the specified quantity of water was used, then water at 17 C. or colder or larger quantities of water were used. Every case was also examined with water at 47 C. in the manner above given. It need scarcely be pointed out that the position of the head should be kept constant during the entire examination.

One can more easily measure the period of latency and the duration, and obtains better dissociated reactions if one conducts the stimulation of the horisontal semicircular canals in this way than if, as is usually the case, one irrigates the head inclined forwards and then bends it back to 60 degrees. One then obtains more pronounced nystagmus and other reactions but not the small shades of difference in the manner of reacting.

After the patient rested irrigation was done with 50—200 cc. of water with the head fixed in a position 30 degrees forwards for examination of the vertical semicircular canals. Mass irrigation was sometimes used, in which case the quantity of water required to produce rotatory nystagmus represented the threshold value. I used a container with water maintained at a constant temperature by electrical means and hung upon a stand in such a way that the water pressure could be regulated. In this method one tries also to give as little stimulation as possible in order to bring out differences of tonus, form, intensity etc., and dissociated reactions with regard to nystagmus, extremity reactions or vertigo etc.

When strong stimulation is used these differences and dissociations are often completely obliterated. One can only say that the patient displays a vestibular reaction and gets no information as to *how* his vestibular apparatus reacts. It is the qualitative test which gives valuable results in these examinations. One must be careful, however, not to draw any conclusions from small differences in the reactions, for even if the examination technique is as exact as possible there are many large sources of error. One must have proper and clear differences in the manner of reacting or the time, corresponding on different occasions and with different forms of stimulation, in order to be able to say that the condition is pathological. Symptoms or reactions which are to be classed as pathological must be constant in repeated tests within a limited period

of time, and also supported by similar corresponding symptoms or reactions obtained by other means.

In cases where the two sides do not correspond anatomically, it may sometimes be difficult to judge the reactions with certainty. But it should be possible anyway to discover a vestibular tonus difference of central origin, dissociations of one kind or another and other reactions which are characteristic of central vestibular conditions should be able to be observed in any case.

Rotatory tests were conducted with a BÁRÁNY rotating chair where the patient's head can be fixed in various positions. The patient was rotated five to ten times for about ten to twenty seconds with his head inclined 30 degrees forwards, 70—80 degrees backwards and sideways. Nystagmus, deviation and past-pointing with arms, falling tendency and vertigo were looked for after each rotation. The movement of the chair was suddenly stopped in each case. With regard to the quality of the reactions, the examination yielded in the main the same result as with caloric irritation, with a reservation for the difference in the form of stimulation. In this case it is not a question of stimulating one semicircular canal or one labyrinth but the semicircular canals which lie in such a plane that they can react to a given stimulation. To some extent, caloric and rotatory stimulation complement each other because one cannot obtain by calo ic irritation the purely vertical nystagmus which is necessary to verify, for instance, a vertical positional nystagmus combined with vertigo. With regard to extremity reactions, one obtains as a rule the same pathological reaction, but there are exceptions which suggest that the vestibular system is not affected in the same place by the various tests (BRUNNER, 27, KLEESTADT, 46, RUTTIN, 77, 78).

In examining vestibular changes of traumatic origin the rotatory test is important from many points of view. It tells us among other things whether compensation or equalisation has occurred within the expected physiological limits between the two vestibular areas, and as stated before it reveals vertigo combined with a certain nystagmus.

c. Optomotor Stimulation.

Optomotor tests were carried out both with a small "drum" according to BÁRÁNY (2, 10) and with a large cylinder according to BRUNNER (24, 25) which is let down from the ceiling and surrounds the patient. According to RADEMAKER's and TER BRAAK's

(23) investigations one can on the whole distinguish between two different types of optokinetic nystagmus of which one is considered to be of cortical and the other of subcortical origin, which explains why one sometimes obtains a reaction with one form of irritation but not with another. The former nystagmus can be obtained with the little "drum" and the latter with the large cylinder. One cannot always distinguish between them because the cortical factor is always there, but it does not always make itself so strongly felt. I observed that in some cases with dissociated vestibular reactions corresponding extremity reactions were obtained with optokinetic nystagmus of subcortical origin but not with nystagmus of cortical origin. The same applies to the falling tendency, and I have therefore always examined the patient standing in the cylinder as in Romberg's test. I then test for nystagmus, deviation and falling tendency in turn with the large cylinder. One obtains the impression that the pathways involved in vestibular and subcortical optomotor nystagmus and in extremity reactions are related to each other — perhaps in some cases they are common. It was not always possible to prove any certain correlation between the result of an optomotor examination and a tipping table examination but it probably exists, since dissociated vestibular reactions including pathological extremity reactions can sometimes be obtained with optomoter tests. This is the reason why optomotor irritation was always employed as a method of examination and it is certain that there is much of value to be obtained from it.

d. Examination on a Tipping Table.

Walking with eyes open and bound was used as a test of kinetic equilibrium, but the results of this examination are difficult to judge objectively and a method of obtaining a better and more objective idea of the patient's disturbance of balance was therefore sought for. One can frequently not draw any certain conclusion from a pathological tendency in walking unless there are other corresponding pathological vestibular reactions. Examination on a tipping table has to some extent proved useful as a complement to, and check on the walking test. The reactions do not direcly correspond apart from the fact that they are both of kinetic origin, but there exists a certain correlation between their appearance as symptoms of a vestibular organic disorder which will be discussed more closely at a later stage.

A specially constructed table, shown in figure 1, was used in the tipping examinations. The surface can be tipped to 45 degrees on all four sides and the axis of the movements lies in the plane of the surface in longitudinal tipping and immediately below it in transversal tipping. A soft rubber mattress, designed to prevent the patient from sliding, is fixed to the surface. In order still further to prevent this, supports are fixed under the mattress on the surface of the table beside the places for the hands and knees. Owing to his own weight the patient sinks down towards these supports and thus stands more steadily, i. e. he does not slide so easily when the table is tipped to its fullest extent. A head rest can be placed at one end for use when examining positional nystagmus and positional vertigo.

During an examination of the tipping reactions, the patient is placed on hands and knees on a line running down the centre of the table parallel with its longitudinal axis of movement. His arms should be straight or slightly bent and parallel and he should rest the palms of his hands on the table. His knees should be separated to the same extent as his arms, and his arms, trunk and thighs should form with the surface on which he is standing as rectangular a figure as is anatomically possible. The head should be somewhat raised and the eyes bound. Before the examination, the patient should be told what it involves and what is expected of him i. e. that he is to try to preserve his balance on the table. He may bend and stretch his arms as he wishes but not move his hands from their original position. He should not tense or strain himself during the examination, for it exhausts him unnecessarily without helping him. After unlocking the table top one can feel at once if the patient is in a normal position and small movements show one almost at once how the patient is going to react. A normal patient makes no opposition to the movements, one does not feel him however rapidly the table is tipped. Normal cases are therefore very easily examined. When the patient has thus been placed in a normal position on the table, an assistant is stationed on the other side to receive him if he falls. Then one tips rapidly to one side and returns slowly to the normal position where one waits for different periods between the movements and tries to distract the patient, making a rapid movement now and again. These must take the patient suddenly and by surprise. He should not therefore know in advance to which side the movements are to be made. Actually this does not signify very much because the movement is

so rapid that he cannot do anything consciously. The period of latency for the extremity reactions is very short. After the rapid movement a few slow movements are made, and it often happens then that a patient who reacted to the former with promptness and the utmost use of his extremities for the defence movements now displays no reaction until the tipping of the table top reaches such an angle that the displacement of the centre of gravity is felt. Often the reaction is then jerky even if the table movement is even and slow.

After these movements through the patient's longitudinal axis the same manoeuvre is carried out through the transversal axis. Here also one must try to have the patient stand in such a way that his weight is evenly distributed in front of and behind the axis of movement. His toes must not protude beyond the edge of the table, since this can upset the results.

The patients have also been examined in a sitting position, placed immediately above the axis of movement. This examination is, however, of less importance from a vestibular point of view for it does not yield reactions which correspond to those obtained with other vestibular tests. The equilibrium reactions of the trunk, for instance Bárány's girdle reflex, do not coincide with reactions on the table and one often finds that a patient who displays a pathological tipping reaction when examined on hands and knees and vestibular reactions in accordance therewith, displays normal defensive reactions in the sitting position. This also appears from Zador's (102, p. 126) investigations. My investigations therefore indicate that tests in a sitting position are of minor importance in examinations for the oto-neurological state. In order to be able to say for certain that vestibularly induced extremity reactions are pathological one must be able to obtain these reactions in different ways, i. e. by employing different forms of stimulation, and to obtain a definite correlation between the manners of reacting. This is the case when the patient is examined on hands and knees but not in a sitting position.

The pictures in fig. 1 show how a normal patient reacts to rapid tipping of the table.

Rapid tipping through the patient's longitudinal axis, for instance to the right, causes a straightening and abduction of the right arm and bending and adduction of the left, thus bringing the body over to the left. There is a corresponding abduction and adduction, though to a lesser degree, in the hips. One can sometimes palpate

Fig. 1. A and B show the normal tipping reaction around the longitudinal axis during $^{5}/_{16}$ of a second, C and D the normal tipping reaction around the transverse axis during $^{7}/_{16}$ of a second.

a relaxation of the adductor semi-tendinosus and semi-membranosus muscles on the right side and a corresponding violent contraction of the these muscle groups on the left. This muscle play can

sometimes be seen in thin people. There is increased tonus and contraction of the right triceps and deltoid muscles and a corresponding reduction on the left side, together with a tonus increase and contraction of the adductor pectoralis major, biceps, and other muscles on this side. The head is twisted slightly to the right and thus retains its position in space.

As a rule, the extremity reactions are the same to both sides, and if the arms are bent, the trunk moved to one side, the head inclined towards the shoulder etc., when the table is moved to one side, then the same movements will occur when there is tipping to the other. The manner of reacting is always stereotyped and identical in normal cases, and the reactions on one side are a reflection of those on the other.

The defence reactions are similar in slow movements but sometimes occur in stages, and the extremity reaction is usually not so comprehensive as in rapid tipping.

Rapid tipping through the transversal axis yields different reactions to backward and forward tipping. — In the former case the hips are extended and the legs and the feet are pressed against the table. The arms are slightly stretched or bent. The body is inclined forward and the head slightly raised. When the table returns to its original position, the patient does likewise. — In the latter case the arms are stretched out from the body, the head is bent slightly downwards, the hips are bent at the same time as the legs are lifted slightly from the table. It looks as though the patient were going to sit on his heels. After the movement, the patient resumes his original position when the table returns to normal. The reactions to slow tipping are similar.

When normal patients are examined in a sitting position their reactions are rapid and stereotyped and the same to both sides. The patient is placed at one end of the table, sitting straight over the axis of movement with his legs hanging freely and his arms resting in his lap. Rapid tipping to the right, for example, causes a deviation of the trunk and head to the left and the latter is slightly twisted towards the left shoulder. At the same time the right leg is extended at the knee and hip joints and the left is strongly flexed at the same joints. All patients do not react in the same way. One often sees a more or less pronounced rotation of the body towards the raised side at the same time as the deviation, sometimes the patient bends forward at the same time, and so on. The

main thing, however, is that the reaction should be the same to both sides, which it always is in normal cases.

As I pointed out before, not much benefit is to be derived from examinations in other positions than on hands and knees, and in this position tipping through the patient's longitudinal axis is the most profitable. The reactions to this latter tipping make it possible to prove a correlation between a pathological tipping reaction and pathological extremity reactions to the other ordinary vestibular tests. Tipping through a transversal axis yields pathological defensive reactions of the same origin but also in cases of positional nystagmus. But this form of nystagmus is combined with vertigo and generally vertical and the patient's difficulties are more due to vertigo than to defective muscle reactions. — Examination in a sitting position usually yields a poor defence reaction if one is obtained with the longitudinal method, but some patients, particularly older ones, display normal reactions. The reaction is more pronounced in cases of cerebellar complications to vestibular defects with acute ataxia, assynergia, etc. All this will be dealt with more fully later.

When the first experiments were made with the tipping reaction as a method of examination for vestibular conditions at the Serafimerlasarett, a table which could be lifted on either side was used. The results were unsatisfactory, however. In the first place one could not take the patient sufficiently by surprise and in the second place this method of investigation did not provide the rotation of the object which was desired, but rather a mere lifting of one side and displacement of the centre of gravity. It was mainly to the latter that the patient reacted. The first cases examined were ones of cerebral tumours and nervous diseases of various kinds with or without vertigo. Towards the end of 1934 my chief at that time, Professor C. O. Nylén, inspired me to continue the investigations and rendered me valuable assistance both with his great knowledge and experience of vestibular questions and by the friendliness and interest which he displayed. I also received literature from him. At about the same time we had the pleasure of a visit at the hospital of Professor A. de Kleyn, who after seeing our experiments had the kindness to send us a model of his tipping table on his return home. All this was encouraging and resulted in a closer examination of the tipping reaction investigations which had been carried out and an attempt to penetrate more deeply into these problems on this basis. To begin with, a

number of experiments were carried out on animals to try to arrive at a method of investigation — a table which was suitable. Since DE KLEYN'S table involved a displacement sideways which complicated the rotatory movement which was desired, my examination table was not constructed on this principle.

The experiments which I have conducted on animals have not been particularly comprehensive and have actually only been designed to reveal the best and most suitable method of examination, that is to say I have tried various methods and ways of tipping. Cats subjected to bilateral or unilateral labyrinthectomy were used. The animals were examined at regular intervals in three different ways according to the methods used for clinical purposes by various writers. The three methods were: tipping upon a surface raised or lowered through an axis in one edge; examination on a table mounted upon two semi-circular rockers, and finally examination on a table where the axis of movement lay in the plane of the table-top and where the animal was placed with its longitudinal axis coinciding with the axis of movement, or transversely to it in the middle. These various methods of investigation involve movements which differ considerably from each other. In the first case one gets a vertical raising or lowering of the object, in the second a displacement sideways. Both methods have an undesirable effect upon the object because there is a movement of the centre of gravity instead of the pure rotation which the examination aims at. Proprioceptive and extroceptive reflexes play an unnecessarily large rôle, making the reaction complicated and difficult to judge as an equilibrium reaction arising only from the semicircular canals. These examinations show also that a cat displays compensation at an earlier stage when examined according to the former method than when it is examined on a table with its axis of movement running through the plane of the table-top and placed in such a way that the length of the cat coincides with and runs directly over the axis of movement or runs transversally across it in the middle. The difference has proved quite considerable in some cases, particularly in the case of large and heavy animals where the gravity factor is more pronounced. Since the table's axis of movement lies a long way from the animal's, the latter is thrown from side to side. The pictures show more clearly the difference in the movements.

A bilaterally labyrinthectomised cat cannot preserve its equilibrium on any of the tables four months after the operations, but

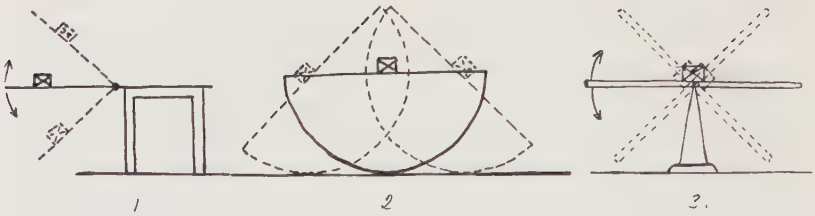

Fig. 2. Schematic drawing illustration the types of tables used and the different ways in which they shift the subject.

it falls in different ways. On the first two table types he is pushed to one side, particularly with type 2, but on the latter he is rolled around. The animal's passive movement is not the same on any of the tables. The difference appears more clearly where unilateral labyrinthectomy has been carried out. Compensation occurs after different intervals on the different tables. Thus the animals display extremity reactions at an earlier stage when examined on a table of type 2 than on one where the axis is as high up under the animal as possible. If compensation after the operation has reached such an advanced stage that the animal reacts comparatively well to rapid tipping of type 1 tables, then the extremity reactions correspond to those which we usually obtained in examination on type 2 at an earlier stage in the development of the compensation. If, however, the animal is placed nearer to the axis of movement and parallel to it on a table of type 1, it reacts less well. The further it is placed from the axis of movement the better the equilibrium reactions become. Thus these simple experiments showed that the different methods of examining extremity reactions were in these cases more sensitive the closer one came to the axis of rotation. A table was therefore constructed which could be tipped around a horisontal axis running through its centre (table type 3). The animal was placed on this table immediately above and parallel to the axis of movement. During the compensation which ensued, it was found that one could observe the progress of this compensation for pathological extremity reactions longer by examination of this kind than with the earlier methods 1 and 2. Thus the experiments indicate that one should try to arrive at a movement which is as purely rotatory as possible and does not involve a horisontal or vertical displacement of the centre of gravity. The latter was found to complicate the examination and to facilitate the animal's ability to react, since

factors other than pure semicircular canal reflexes asserted themselves in a more or less pronounced form. The displacement in the examination complicates the reactions and make it impossible to say with certainty how they are caused since different forces contribute to upset the equilibrium. The movement of table type 3 meets more closely the object of examination, i.e. a rotation through the animal's fronto-occipital and bitemporal axes without any major horisontal or vertical displacement. The animal's reaction then becomes a purer vertical semicircular canal reflex with the least disturbances from other areas which contribute to the preservation of equilibrium. The extremity reactions can also be studied more easily since the animal is in a more stationary position. — — — Altogether ten experiments with animals were performed. Three animals were bilaterally labyrinthectomised and seven unilaterally — three on the left and four on the right side.

The table which has been used in the examination of the present series is constructed on the above principles, and the same principles have been followed during the actual examination. That is to say I have attempted to arrive at a movement which is as purely rotatory as possible without any displacements of the centre of gravity to complicate matters. RADEMAKER and GARCIN used a table built in the same way in which, however, the rotatory movement was suddenly stopped. This was done as long ago as 1929, but in 1933 (73) they considered that the examination could be done equally well on a stretcher or a bed which was lifted at the sides. The reason for their return to their first method of examination was that they obtained the impression that the patient became frightened, tense, etc., when the table was used and furthermore showed a greater tendency to slide on the table than in for instance a bed. My series is of course of a very different nature and more easy to examine and I have not noticed that our method of examination has caused any psychical strain on the patient.

DE KLEYN and VERSTEEGH both used a "cradle" for their examination but have also made use of RADEMAKER and GARCIN'S table, particularly in one case where they particularly mention this fact. The reactions were the same on both tables, but it is evident that the writers thought that the vestibular functions were not affected in the same way by examination on the two tables.

With ZADOR'S table, a movement can be made at a long distance from the patient. He attaches importance to this because the

table's direction of movement takes the patient by surprise. This is doubtless correct and important, but one can obtain the same effect with tipping by hand in the manner which I used. I also tried suddenly stopping the table movement but did not find it satisfactory because the patient reacts more easily to this jolt than to a slow stopping of the table movement. One might think that because sudden braking is applied in making rotatory tests on a Bárány chair, the same should be satisfactory here also. Both cases involve rotation. The difference is, however, that tipping involves a displacement sideways of the centre of gravity of a large mass, the equilibrium of which depends upon other factors than labyrinthine and vestibular functions. These factors play a larger rôle in sudden than in slow braking. The reaction becomes abrupt and one does not obtain the shades of difference in the reaction which in some conditions are its particular feature. This applies particularly to patients with slight ataxia who, when there is rapid braking, fall over to the lowered side of the table, but when the stopping movement is slow frequently display a falling tendency towards the raised side with clear hypermetria, assynergia, etc. This form of reaction is not unusual, but it does not appear unless one can analyse the final phase of the reaction. Over-sudden and violent braking of the tipping movement obscures the characteristic features of the final phase. This is important in a differentiated equilibrium examination and increases the value of the tipping examination from the standpoint of topographic diagnosis.

The tipping should begin rapidly, but not jerkily so that there is a rapid displacement of the centre of gravity. If the patient is properly centred on the table this need scarcely be feared. The threshold value for the semicircular canal reaction is thus reached at once, and the defence occurs automatically. If one begins the movement slowly and accelerates it one does not obtain the first phase of the defence movement as a reaction of vestibular origin, which it is intended to be. The second phase is passive and the displacement of the centre of gravity makes itself felt. The third is the extroceptive and proprioceptive, with correction movements originating from the contact of the supporting surfaces with the table-top and the deep sensibility in muscles and joints as the principal motive forces. Ataxias of cerebellar origin make themselves felt in this latter phase on condition that the movement is not violently broken off so that the finer shades of the extremity

reactions in the final phase are levelled out and obliterated. For the object of the tipping test is not merely to ascertain whether the patient can stand on the table during rapid tipping but primarily to show *how* he reacts. That is to say that the tipping examination does not only indicate whether or not the patient is capable of certain vestibular-cerebellar reactions but also provides a differentiated picture of these reactions with particular regard to certain cerebellar functions. This is the most interesting feature of the method for it can, and certainly will in time be developed just in this respect. It is a differential diagnosis that one wants, and this method offers the possibility of obtaining one in respect of pathological extremity reactions during a disturbance of equilibrium of vestibulo-cerebellar genesis. This matter will be dealt with more closely later on.

Since it is not necessary when conducting a tipping examination in the way given here to take so much into account the second phase of the reaction, I have found it sufficient in practice to reckon with two phases. These are:

1. The "initial" or "vestibular" phase which expresses a vestibular reaction — the kinetic adaptation. It is better to use the word "vestibular" than "labyrinthine" since the reaction does not only depend upon the functional condition of the peripheral sensory organ but as much upon its central connections and centres. A central injury which affects the vestibular tracts or pathways for the inducement of extremity reactions is very usual after head injuries. Most frequently it is then not possible to prove the existence of any peripheral or labyrinthine disorder.

2. The "final" or "cerebellar" phase which expresses the symmetry, coordination, etc. of the induced extremity reactions and by which these latter are coordinated into well-balanced and functional defensive reactions. It represents the static adaptation to the new position and is particularly prominent when lesions in the cerebellum or brain stem affect the vestibulo-cerebellar tracts or pathways for extremity reactions.

From a practical and clinical point of view, these are the two component parts of the tipping reaction which, particularly in the case of pathological reactions, give it its character. The more damaged one component is the more it sets its mark on the reaction. This will be dealt with more closely later on.

The position of the head does not appear to be of any importance

in the reaction. Following RADEMAKER (71, p. 128) I have examined both normal and pathological cases with the head in such a position that the horisontal semicircular canals are stimulated, that is with them parallel to the plane of rotation, and in such a position that the vertical semicircular canals are parallel to the plane of rotation, but I have not discovered any difference in the nature of the reaction. This distinguishes our reactions from those of animals, for the latter display no extremity reaction to rotation through a cranio-basal axis (RADEMAKER, TAIT, etc.). It is difficult to explain the reason for the difference in the reaction of the human and animal labyrinth, but presumably it is to be ascribed to the difference in stato-kinetics which exists between human beings and animals -- and to the need for better reflexes in the semicircular canals when the gait is upright and the position of equilibrium more unstable. It is in point of fact the central connections for extremity reactions which have been developed and improved for the preservation of equilibrium in humans.

In some cases the patient was tested in a diagonal position on the table during tipping in accordance with TAIT and MCNALLY (91) but it did not yield better reactions than ordinary tipping through a longitudinal axis. Experiments on animals are tempting but they can evidently not be applied to human material.

In order to make it easier to judge the reactions on the tipping table, we have in some cases filmed them, but in the long run this is too costly. Nor is it necessary for judging the cases. One can see on the film strip both the table movements and the reactions of the patient and one can work out how rapidly the tipping has been executed and obtain an idea of the period of latency for the reactions in pathological cases. It is particularly in the final phase of a pathological reaction that the ataxia appears clearly on the film. Normal cases react like lightning to the movements on the table and one cannot in this way measure the period of latency. The table movements should be made in five to seven sixteenths of a second in the examination of pathological cases, but in examining normal cases one can of course tip as fast as one likes. As a rule it is safe to say that tipping faster than about half a second requires vestibular reactions for the preservation of balance. This applies to tipping through the patient's longitudinal axis. A couple of patients with both labyrinths dead for a long time were examined and displayed no real defensive reactions to tipping faster than that last mentioned. They reacted normally, however, to slower

tipping. The experiments do not constitute a direct proof, but the observation regarding the speed of the table movement agrees closely with the experience obtained from examining the pathological cases in this material. They show how important it is that the tipping movement should be made at sufficient speed.

D. The Application of the Method and the Pathological Signs Observed.

In the preceding sections I have dealt with the various tests used in the otoneurological examination of my cases of post-traumatic vertigo. This has been done to show how the patient is examined and to try to prove whether a relation exists and, if so where, between the various vestibular tests included in this method, with particular reference to the tipping test. With regard to the latter, different reactions and manners of reacting which are characteristic of normal cases have been mentioned. I shall now give a brief description of the signs and symptoms observed with the same battery of tests which appeared to be most characteristic of peripheral or central organic injury in the eighth cranial nerve with its various connections, particularly with a view to trying to prove a correlation between pathological reactions on the tipping table and other pathological vestibular reactions.

It is not possible to draw any certain conclusions from the spontaneous signs alone. It is only spontaneous nystagmus which can here be regarded as a purely objective sign of organic injury, but by itself it does not tell us much. It is the induced signs and reactions which are of decisive importance, particularly where different forms of irritation are found to release corresponding reactions.

Of the induced signs and reactions already mentioned, positional nystagmus and nystagmus induced by head rolling (head movement test) have previously been described in respect of their type, occurrence and importance to diagnosis, and there is therefore nothing to add at this point. Caloric, rotatory and optomotor stimulation and examination on a tipping table do, however, in some cases yield signs and reactions which in special circumstances can be certain indications of organic injury in the eighth cranial nerve and its connections.

1. Caloric Stimulation.

With the use of the method previously described, the following reactions can generally be observed in pathological vestibular post-traumatic cases:

a. The reaction typical of a purely peripheral labyrinthine injury.

b. A pathological nystagmus response which cannot be grouped with other nystagmus reactions. ("Abnorme Schaltung, perverted nystagmus, détournement du nystagmus".)

c. Vestibular tonus difference of central origin according to KOCH.

d. Dissociated vestibular reactions.

e. Eye deviation = "Bulbabweichung" according to STIER and BEYER.

These can occur as an isolated phenomenon but one usually finds, in cases of central vestibular injury, more than one of these pathological reactions at the same time in the same patient. This applies usually to the last four manners of reacting which can be classed as indicators of central vestibular disorders, on condition that corresponding reactions are obtained during further vestibular tests. The test is not of course physiological — it has great failings, however carefully it is carried out — and when a pathological reaction is suspected one ought therefore to look for verifying reactions. This is merely a precautionary measure for judging the case. One should make comparisons with other reactions, after rotation and optomotor stimulation and in tipping tests, in their relevant parts, and consider hearing, etc.

a. *Labyrinthine* destruction of a purely peripheral nature yields no caloric reactions, and a corresponding hearing defect, i. e., deafness. Peripheral hypersensitivity and hyposensitivity, which can be determined by measuring the duration and period of latency, can be regarded as pathological if they occur unilaterally and if the time difference is definite and constant. If it is bilateral and the same on both sides, it may be, and usually is, physiological and one cannot, therefore, judge its importance as a pathological reaction. In practice, therefore, it is the difference between the two ears' sensitivity and ability to react to cold and hot water which is decisive, as in the following example of nystagmus:

Right ear		Left ear
⟶	10 cc. 27 C. water	⟵
5—200 sec.		10—120 sec.
⟵	10 cc. 47 C. water	⟶
10—150 sec.		20—90 sec.

In this case we have a short period of latency and long duration in the right ear. Peripheral hyposensitivity for instance on the right side gives a long period of latency and a short duration in the same way.

Examination with cold and hot water must yield corresponding reactions and one should carry out an extra examination in order to check the result. Furthermore, hearing is often modified in a similar way. It is not often that one finds purely peripheral damage, as above, after head trauma, but it is usually a condition following inflammatory modifications in the middle ear. Frequently it is difficult to decide the connection with the trauma.

b. *Pathological nystagmus response* signifies here both a nystagmus in an unexpected direction after irrigation, on condition that the original head position is maintained, and a change in rhythm, intensity, form, and frequency under the same conditions. This disorder is usually of central origin. In the former case one may get an inverse or perverse nystagmus (paradox nystagmus) (DE KLEYN and STENVERS, 48) and then usually oblique or vertical. In the latter case the nystagmus may beat in spasms with long intervals between them. The nystagmus seems to get caught up and sometimes one finds a fine, high frequency nystagmus alternating with longer beats. This is obtained by stimulating one side while normal nystagmus is obtained on the other, but one can also obtain different forms of pathological nystagmus response in the two eyes from one stimulation. Consequently one must compare the nystagmus in the right and left eye after each irrigation in order to observe whether the eyes receive the same nystagmus impulses and also to discover whether irritation of the right and left vestibular nerves yields different nystagmus responses. In both cases, the nystagmus must in certain conditions be regarded as of central origin and due to changes in the vestibulo-ocular pathways of the brain stem. According to DE KLEYN (47), both peripheral and central injury can yield abnormal nystagmus response. The intensity can reach any degree up to nystagmoclonus when it is raised to the utmost. In these latter cases there is a dissociation of the

vestibular reactions since the extremity reactions and the vertigo are not equally pronounced. Berger (19) and de Kleyn (47) point out that the secondary reactions are often reduced and Frenzel (37) points out with particular emphasis that the intensity of the nystagmus is often pathologically changed after irrigation and that this is an important object of study when judging the vestibular functions.

c. *Vestibular tonus difference* of central origin means according to Koch (53—56) a condition with excessive nystagmus to one side both in the matter of period of latency and duration. The intensity and the form are often changed at the same time, however, regardless of which ear is irrigated. On the whole the same reaction is obtained both to caloric and rotatory stimulation. This "Nystagmusbereitschaft" was known long ago thanks to experiments on animals by Dusser de Barenne and de Kleyn (14). First Vogel (96) and later Koch (53—56) among others described the same phenomenon in human beings after bilateral irrigation according to Fischer and Ruttin. One comes across this fairly frequently in examining cases with posttraumatic vertigo and it is considered to be a sure sign of central vestibular injury (Koch, 53—56, and Morsier and Barbey, 61). Frequently these patients display no spontaneous nystagmus reactions. We were only able to obtain a "Kopfschütteln"-nystagmus in exceptional cases. The pathological and anatomic basis for this manner of reaction is not known but it is thought that the disorder is located in the area within the brain stem where the vestibular impulses of peripheral origin are distributed through the posterior longitudinal bundle to the eye muscle nuclei to produce nystagmus. Expressed in the same way as under a. the vestibular tonus difference looks like this example.

Right ear		Left ear
⟶	10 cc. 27 C. water	⟵
5—200 sec.		10—120 sec.
⟵	10 cc. 47 C. water	⟶
20—90 sec.		10—150 sec.

In this case, there is a nystagmus increase to the left on irrigation of both the right and left ear. The period of latency is reduced and the duration increased. Since the intensity and frequency are also as a rule increased, the difference in the nystagmus appears even more pronounced.

Spontaneous and positional nystagmus with the same or similar direction as in "Nystagmusbereitschaft" also occurs. One then obtains the impression that this nystagmus is made manifest by vestibular stimulation. We can see examples thereof in the present series and this circumstance has also been demonstrated before (Van den Boorn, 22, Mittermaier, 59).

d. *Dissociated vestibular reactions* to caloric and rotatory vestibular stimulation are fairly usual in these cases. One obtains a dissociation in respect of the reactions which can express itself in many ways according to the injury to the various vestibular central tracts and pathways. These are not known in full. But on the whole it is safe to assume that there are separate tracts and pathways for the horisontal and vertical semicircular canals and that the pathways for the nystagmus reaction are separated from those which convey the impulses for extremity reactions of vestibular origin (Isaac Jones, 45, Eagleton, 32). In such a case the impulses travel from the sensory organ, the labyrinth, to the vestibular nuclear areas in the medulla oblongata and pons with separate tracts for the horisontal and vertical semicircular canals. A distribution then occurs in such a way that the impulses for eye movements are forwarded as mentioned above while the impulses for extremity reactions are transmitted through the cerebello-vestibular nuclear areas to the brain cortex, with separate pathways here also for the horisontal and vertical semicircular canals. Here the muscle reactions necessary for the preservation of balance originate. In order to serve their purpose as satisfactory equilibrium reactions they later require coordination, symmetry, synergia, etc. all of which are cerebellar functions. A stimulation of the horisontal semicircular canal causes an impulse which can be seen in a movement of the eyes, in extremity reactions and in vertigo. The same applies to the vertical semicircular canals, although one cannot stimulate them individually but must be satisfied with the reaction one obtains and judge whether it is pathological from one's experience of normal cases. The same applies largely of course to the examination of the reactions of the horisontal semicircular canal since the caloric test is really not a physiological form of vestibular stimulation. One provides an impulse and studies its results and has only to judge the condition accordingly.

Equally little is known for certain about the pathway for vertigo. Jones, who suggested the rough scheme briefly summarised in the

foregoing for the vestibular tracts and pathways as long ago as 1918, only really reckons with two, one for nystagmus and one for vertigo. According to this theory the extremity reactions are a result of the vertigo. "No extremity reaction without vertigo" was his motto. This is not absolutely correct, however, for one can find pathological vertigo at the same times as normal vestibular extremity reactions, and vice versa. That is to say, the dissociation can also apply to vertigo in relation to the two other reactions which are obtained in cases of induced vestibular tests, i.e. the eye movement and the extremity reaction.

The dissociated vestibular reaction is a phenomenon of purely central origin for it involves a partial injury of the vestibular function with modifications and absence of certain parts of it at the same time as hearing is usually intact. One can scarcely imagine that any division of the partial functions in a vestibular respect can occur in the peripheral sensory organ — the labyrinth. On the other hand, partial modifications of the vestibular functions can occur if the central vestibular tract and pathway above the place of entry of the vestibular branch into the medulla oblongata and after the subsequent division into various pathways, are damaged, as well as in cases of damage to the vestibular nuclear areas in the medulla oblongata, pons or cerebellum. JONES and FISHER claim that they can pathologically and anatomically verify their assumptions about the vestibular central connections in some cases. BARRÉ (15, 17, 30) and his pupils have in the matter of the dissociated extremity reactions to ordinary vestibular tests, gathered these together under the name "syndrome vestibulaire dysharmonieux". A number of cerebellar disorders were examined and in these cases BARRÉ has been able to demonstrate a deviation in the vestibular reactions when compared with normal cases and with purely peripheral labyrinthine disorders and that the difference in the reaction lies in a partial change in the vestibular functions — a disharmony in the vestibular reactions — with pathological extremity reactions and at the same time intact, normal eye movements of vestibular origin. In these cases we have one example of what can constitute a dissociated vestibular reaction, but the incongruency between the induced vestibular reactions can assume many forms and the disharmonious vestibular syndrome is only one of many. For instance one can find a pathologically increased nystagmus frequency (such as nystagmoclonus) without any corresponding change in the vertigo and the extremity

reactions. In other cases the vertigo is pathological while the other reactions are normal. These pathological phenomena are tied to certain definite vestibular areas. That is to say they can only be caused by irritation of certain definite areas in the vestibular system. They are anatomically and functionally fixed to the horisontal and vertical canals and their pathways on one or both sides.

A dissociated reaction in the matter of nystagmus occurs if the vestibulo-ocular pathway between the vestibular nuclear area and the posterior longitudinal bundle is damaged or if the injury is situated farther up on the connections for nystagmus (Metzger, 58). One can call all this the "nystagmus tract" and the disorder a "rising dissociation" according to Cambrelin (28). One obtains a corresponding reaction in respect of pathological extremity reflexes if the vestibulo-cerebellar pathway between the same nuclear areas and the nuclear areas in the cerebellum, or the fibres for the same extremity reactions here or above these areas are injured. One can class these altogether as the "extremity tract" and the functional disorder as a "descending dissociation" according to Cambrelin. Less is known about the "vertigo tract". The reactions here involve a perception of upset equilibrium — a feeling, a mental experience which cannot be measured like other reactions since it constitutes a purely subjective reaction liable to great constitutional variations. — These dissociated reactions I have now described occur comparatively often after head injuries and are of great diagnostic value owing to their great objectivity. Every case of head injury which complains of vertigo should therefore be examined for these dissociated reactions. The dissociated vestibular reaction, embracing pathological extremity reactions, is of course the one which is of the greatest interest for the purposes of this work for it shows a correlation with a pathological tipping reaction.

The reason for the pathological reaction is a disorder in the posterior cranial fossa which injures the vestibulo-cerebello-cerebral pathways either on the same side or on the opposite side, the normal function of which is to release the reflexes or reactions which are designed to maintain equilibrium by extremity movements. Barré, Charbonnel, Alfandary (15, 17, 30) and others attribute importance to these reactions particularly for detecting disorders in the cerebellum. One can

obtain with their help an early diagnosis of a cerebellar disorder which otherwise yields no clear signs since the subjective troubles are often interpreted as being of purely vestibular origin. Injury to the vestibulo-cerebello-cerebral pathways, which need not yield any spontaneous signs, becomes manifest as a result of vestibular stimulation which brings out extremity movements of vestibular origin which are then in one way or another abnormal. A "disharmonious vestibular syndrome" arises consequently as a result of a cerebellar disorder which affects the vestibular connections for extremity reactions, for the pathways pass through the cerebellum and the reactions are closely linked with and dependent upon the functioning of the cerebellum for the coordination, etc. of the reflexes into useful, well-balanced movements. Caloric and, in its relevant parts, rotatory stimulation bring out the pathological reactions in the form of abnormal deviation, past-pointing and a certain falling tendency at the same time as the nystagmus and vertigo reactions are normal. The examination is carried out as previously described and proper, clear reactions repeated during various tests must be obtained before one can be certain that the reaction is pathological. Usually one finds that the deviation and pointing are reduced, absent or crossed on one side, as a rule on the same side as the injury if it is situated in the cerebellar hemisphere or nuclei on this side. Injury in the cerebral cortex gives a pathological reaction in the extremity controlled by the area concerned. Injury involving a breakage of the vestibulo-cerebello-cerebral connections causes deviation and pointing error just like a purely peripheral destruction of the labyrinth.

The pathological reactions can be occasioned by stimulation of the vertical semicircular canals while normal reactions are obtained from the horisontal semicircular canals, or vice versa, on the same or both sides. If they occur uniformly on both sides one must consider the possibility of their being constitutional. The anamnesis and symptoms in general often clarify this, however.

It is not known for certain what the effect is of injury in the peduncles (corpus restiforme, brachium pontis and brachium conjunctivum) but JONES and FISHER (45) claim that in their cases the dissociation resulted in an absence of deviation and pointing on the same side as the injury. This should apply to the nerve filaments through the brachium pontis and brachium conjunctivum, for the latest investigations indicate that the corpus restiforme does not convey any vestibular fibres (LORENTE DA

Nó). According to Charbonnel and Alfandary (30) the whole subject is not yet fully explored.

The essential and most important feature of the whole question of dissociated reactions is that Barré has shown that when the dissociation applies to pathological extremity reactions the reason is to be sought in damage to the vestibulo-cerebellar pathway. In cases of unilateral pathological reactions one must reckon with a cerebellar factor as a contributory cause. If it occurs bilaterally with a total absence of reactions this may be due to a purely peripheral injury or to a central disorder involving a breakage in the vestibulo-cerebello-cerebral pathway for these reactions as a whole.

Bárány (4, 8, 11) assumed that there exists a centre in the cerebellar hemisphere designed for arm movements *inter alia*. Later investigations by Fischer and Wodak (36), Dusser de Barenne (13), André-Thomas (95), Ingvar (44) and others have shown that this is probably not correct. The conditions with regard to dissociated vestibular reactions do not either accord with Bárány's assumption, for deviating and past-pointing are vestibular reactions which can be obtained by suitable vestibular stimulation, and the function of the cerebellum in the reaction is mainly a partial one and serves to coordinate the movements. This is substantiated and demonstrated still further by examination on a tipping table. One must not regard deviation for instance as a reflex but as reaction, for its structure is complicated by an act of will. — A proof that the deviation and past-pointing obtained after caloric stimulation are of vestibular origin exists in their correspondence with a certain form of tipping reaction, for the latter is not pathological except where a dissociated vestibular reaction is obtained in the extremities in the form of pathological deviation and past-pointing. Since the tipping reaction is a reaction of pure vestibulo-cerebellar origin, the deviation and pointing reaction cannot by simple analogy be otherwise induced. This does not prevent the cerebellum from being of importance to the tipping reaction, but from another functional point of view. [Dusser de Barenne (13), Fischer and Wodak (36).]

Cambrelin (28) has dealt in an essay with the disharmonic vestibular syndrome according to Barré and Charbonnel, with particular reference to cerebellar disorders. He points out that the spontaneous symptoms which follow disorders of the hemisphere are in themselves dissociated, with nystagmus primarily to one

side and deviation and falling tendency to the same side. He considers therefore that what these writers call the disharmonious vestibular syndrome is actually a cerebellar hemi-syndrome. In support of his view he cites a number of tumour cases which displayed a disharmonious vestibular syndrome but not induced dissociated vestibular reactions. His inference is correct, but the value of the induced dissociated vestibular reactions is that one cannot without vestibular tests obtain indications of disorders in the posterior cranial fossa in the early stages, when there is vestibulo-spinal disorder. This is the most valuable feature of the examination. He says nothing about the tipping reaction, but one feels that it would be a very obvious test. BARRÉ and CHARBONNEL have not mentioned it either, but ALFANDARY mentions it as a form of vestibular test.

e. *Deviation of the Eyes.* (Bulbabweichung.) According to STIER (87—90) and BAYER (21) deviation of the eyes has sometimes been found in cases of general vertigo after head injury. In 1937 they examined 200 cases and found it in 16 of them. One finds a deviation of one or other eye after caloric and rotatory stimulation. The eye moves in the normal way to the side when the patient looks in the direction of the nystagmus. After a while the eye begins to beat, does not look in the same direction as the other but tends back to the middle. The nystagmus comes in spasms, while the eye slowly returns to the direction of the gaze. It is not known for certain to what this phenomenon is due. It may be assumed either that the vestibular phase is more strongly pronounced in that eye or that there exists a latent strabismus which is brought out under BARTHEL's or FRENZEL's spectacles. Experience shows, however, that one does not obtain this movement clearly without caloric or rotatory stimulation. The reaction, therefore, seems to have some connection with vestibular eye functions. It seems as though there is a latent weakness in the eye muscle nuclei with regard to the transmission of similar impulses to corresponding muscles, and that this weakness is brought out by vestibular stimulation. The phenomenon is ceaselessly repeated during the test, and the nystagmus which appears varies in amplitude and intensity. At the same time there often occurs a dissociated reaction. The patient's symptoms in cases of experimentally demonstrable eye deviation are typical. They often find it difficult to read for a long period or to carry out work which strains the eyes. They often complain of vertigo inasmuch as objects are inclined

to merge together and become misty and unclear. The dizziness is frequently of opto-kinetic origin. Angio-neurotic troubles are often strongly pronounced. The eye deviation is also brought out by optomotor stimulation, particularly in a cortical form, and often one finds the same abnormal nystagmus and the same subjective troubles as after caloric and rotatory stimulation.

2. Rotatory Stimulation.

Dissociated reactions, particularly in the extremities, are equally, if not more clear after rotatory than after caloric stimulation, with the result that there is frequently a clearer correspondence between the rotatory and the tipping reactions. This may perhaps be because both these reactions are rather physiological. In addition the rotatory test complements the caloric in examination of the vertical semicircular canals, but one cannot obtain any vertical nystagmus by caloric stimulation. In cases of vertigo arising from definite positions of the head and body and linked with vertical nystagmus, one can sometimes, if the patient is rotated so that a corresponding nystagmus occurs, obtain the same vertigo symptoms as well as a pathological nystagmus of prolonged duration, an irregular nystagmus of a different kind and pathological vegetative symptoms. These symptoms reappear during repeated tests and one can thus confirm the fact that a vertigo is of organic origin and linked not only to position but also to vertical nystagmus. Since the nystagmus combined with vertigo is pathological in these cases, everything indicates that the vertiginous ailments are of vestibular, central organic origin. Vertical nystagmus has only been taken as an example. The same also applies of course to vertigo linked with rotatory nystagmus. — The vestibular tonus difference of central origin, the dissociated reactions, the pathological nystagmus response and the eye deviation are on the whole produced by rotatory tests to the same extent as by caloric stimulation, and this examination is therefore valuable as a check. All this is subject to the reservation that the two tests are physiologically different.

3. Optomotor Stimulation.

Patients with optokinetic ailments sometimes display a poor, uneven and jerky nystagmus to one side and normal to the other on optomotor stimulation. Sometimes one obtains it clearly with

the little drum and sometimes with the large cylinder. If the optokinetic spontaneous disorders are clear, then they are frequently accentuated by stimulation of both kinds. A pathological nystagmus response and a pathological vertigo linked with nystagmus usually correspond better with optokinetic nystagmus of subcortical origin than of cortical origin. The vestibular tonus difference also corresponds better as regards direction with optokinetic nystagmus of subcortical origin. Pathological vertigo linked with nystagmus sometimes corresponds, particularly in cases of vertical pathological nystagmus, and it is frequently also the case that the patient spontaneously reveals this by saying that he recognises his spontaneous dizziness when the object moves in a certain direction. If one then examines the nystagmus response, one finds that it is almost always pathological. All this is more usual when the optokinetic nystagmus is of subcortical origin than when it is of cortical origin.

The optomotor test is as yet very little explored, and clear directions for its clinical use only exist in certain respects. One sometimes finds, however, that one can also obtain a reaction which corresponds with vestibularly induced extremity reactions, One can obtain the same abnormal deviation and pointing reaction with optomotor stimulation of subcortical origin as with dissociated reactions induced by caloric and rotatory means. This suggests that there may be some relation between the pathways for extremity reactions to subcortical optomotor stimulation and those induced by ordinary caloric and rotatory stimulation. The same applies in many cases to the induced falling tendency.

One does not always obtain reactions which correspond, however, and I do not dare therefore to ascribe any importance to this test as far as the dissociated reactions are concerned.

A spontaneous nystagmus can be strengthened or obliterated. If it is of vestibular origin, subcortical optomotor stimulation usually strengthens it. An absence of optokinetic nystagmus may be due to various reasons, some of which can be disclosed during the subsequent tests.

4. Examination on a Tipping Table.

According to Rademaker, the defensive movements during rapid tipping with the patient on hands and knees consist of three phases. Since the method previously described causes only a very

small displacement of the patient during longitudinal rotation, I have tried to make the passive phase of the reaction as small as possible thus bringing out the initial and final phases. One need not therefore reckon here with more than the initial and final phases. It is the defensive movements in these two phases which characterise the tipping reaction in different pathological conditions. In the first phase, defects in the vestibular-labyrinthine reactions are disclosed, and in the latter defects chiefly in the cerebellar reactions. Normal cases display a perfect cooperation between these two phases, the one merging into the other. Defective functioning in one or the other phase causes a typical defensive movement in pathological cases from which important diagnostic conclusions can be drawn. Recent destruction of the labyrinth causes failure to react in the first phase and falling to both sides. If the injury is unilateral and the other labyrinth is normal, then the defective reaction is temporary. If the destruction is bilateral, the defective reaction is permanent. This applies to peripheral injuries. In the case of central vestibular disorders on the other hand, every degree of transition exists between the phases. The more the second phase is modified, the more the pathological tipping reaction is due to a cerebellar disorder. Ataxia, asynergia, assymetr a and hypermetria are then clearly apparent.

Unilateral destruction of the labyrinth gives no defensive reaction and the patient falls as soon as the table is tipped at a certain speed. At first, his inability to preserve his balance is approximately the same to all sides, but gradually an improvement occurs, presumably because the healthy labyrinth assumes to an increasing degree the functions with regard to equilibrium reactions, of the labyrinth on the injured side. Gradually a condition occurs in which the tipping reaction to all axes of movement is as normal as though there has never been any destruction of the labyrinth. The healthy side has given complete compensation. The development of this compensation, from the first attempts at a reaction in the first phase to complete restoration can be observed and studied, and one sees at the same time that the second phase of reaction remains comparatively intact from the very first. One cannot of course see it in the beginning because the patient falls, but at an early stage of the healing it appears in the strikingly good stabilisation which occurs to one side. The patient soon regains his power to preserve complete balance to this side, and gradually one sees the same process repeated in tipping to the other. As a rule the

defective reaction remains longer on the side of the destroyed labyrinth, but not always, for some unknown reason. After unilateral labyrinthitis or labyrinthectomy with elimination of the labyrinthine function, the patient is completely unable to preserve his balance during the next few days even in slow tipping, but this condition is very temporary.

A unilateral loss of the labyrinthine functions of traumatic origin usually takes longer to arrive at compensation, presumably owing to intracranial vestibular injuries. A young person obtains complete compensation more quickly than an older one, and where there are partial injuries it is a very long time before the patient regains his normal equilibrium reactions on the tipping table. It seems as though the little functioning power remaining prevents the development of compensation from the healthy labyrinth. From the point of view of healing, total destruction and loss of function on one side is preferable to partial, for the patient is then rendered invalid for a shorter period and the compensation is surer and more stable.

A common feature of unilateral and bilateral peripheral vestibular injury is that the patient's pathological reaction is most pronounced in the first phase, and no asynergia, hypermetria etc. are displayed. The patients fall without any defensive movements, but their static stability is good with sure and well adapted extremity movements.

As regards vestibular injury of central origin, which is encountered most frequently in cases of posttraumatic vertigo, the tipping reaction usually differs from the reaction yielded by the aforementioned peripheral modifications of the labyrinthine functions. The reactions are affected to different degrees by cerebellar signs which appear in the final phase and sometimes completely dominate the reactions as a whole. As a rule one must expect peripheral vestibular injury after head trauma to be accompanied also by a central injury, and the tipping reaction is therefore rarely unchanged in its final phase. There are exceptions, however, and I have seen one case in which the tip of the petrous bone was broken off without any other changes in the cranial bones and without any pronounced commotio cerebri. After the acute symptoms had ceased, this case yielded the tipping reaction of a peripheral injury with no pathological change in the final phase during the process of healing. In this case complete compensation occurred at an early stage. If the vestibular branch is cut, as for

instance in a case of Ménière's disease, there occurs subsequently a considerable and typical modification of both phases. The first phase gradually improves but the change shown in the final phase remains for a long time and one gains the impression that it delays healing and compensation. It sets its mark upon the reaction for a long time — years — after the operation and the patient has an almost constant feeling of insecurity particularly during rapid movements. Complete healing occurs gradually, and the smaller the injury is to the cerebellum the earlier it occurs for it is the cerebellar lesion which delays compensation in a statokinetic respect.

In view of what has been said before regarding different reactions to tipping table examination, one could divide the pathological cases into two groups, one consisting of the cases whose pathological reactions are primarily of vestibular origin and the other where they are primarily of cerebellar origin. This is not sufficient, however, for the two phases of the reaction are not always so definitely separated from each other in cases of head injury. A purely peripheral labyrinthine disorder without central vestibular injury yields a characteristic reaction, while certain nervous diseases sometimes yield a set of reactions which are almost purely cerebellar in nature. Such cases are encountered rather rarely, however, after head injuries. It is therefore more rational to make the division follow other functional considerations which are also typical of a pathological reaction and in which the displacement of the phases is also brought out and more or less sets its stamp upon the reactions as a whole. Central vestibular injuries are most frequently encountered after head trauma, whereas unilateral peripheral injury as a rule heals comparatively early and is therefore not seen so often. The latter yields no dissociated vestibular reactions and consequently no pathological final phase in the defensive movements to complicate matters. Examination on a tipping table in these cases is principally of importance because one can with its help partially exclude central vestibular injury and also observe the compensation and decide when it is complete, that is to say when the patient's organic vestibular healing is complete in respect of equilibrium functions.

One can distinguish between four different types of pathological tipping reactions when testing cases of post-traumatic vertigo. They are not strictly separated from each other but can merge into one another and, in cases of central vestibular injury, can express

various degrees of injury to the central vestibular areas and their various connections. The more extensive and serious is the injury, the more the reactions are dominated by cerebello-cerebral factors which have not so much directly to do with the vestibular functions but which are made manifest by examination on the tipping table. The tipping shows them up more clearly than any other examination, they immediately attract more attention and they give the whole tipping a characteristic appearance. It is this circumstance which has led to the division into various types, even if they only represent one and the same injury or modification, of various degrees of gravity or central extension.

This classification is not ideal but merely shows the most characteristic feature of the reaction as regards the falling tendency. In each type group, the special reaction in the first and last phases must be observed. This division has been chosen because the patients can be more or less grouped under these four types in cases of vertigo after head injury.

I. Poor defensive reactions to both sides or in all axes of movement with falling tendency more or less pronounced in the direction of the lowered side of the table. As previously mentioned, unilateral destruction of the labyrinth causes this reaction at first only, later one of the next type, after which a normal reaction gradually returns on condition that the other labyrinth can compensate for the loss of function. Bilateral labyrinthine destruction causes a total and permanent inability to preserve balance during rapid tipping movements. In these cases — pure, simple, peripheral vestibular disorders — the first phase of the reaction is defective while the final phase displays no pronounced ataxia. If the final phase is pathological, then there is also a central vestibular injury which it must be possible to demonstrate by some other method in the vestibular examination to be classified as certain. Figure 3 illustrates a tipping reaction of this type in a case of central vestibular organic injury.

II. Poor defensive reactions are more usual to one side — the lowered side of the table. This is either a stage of healing after peripheral vestibular injury with a defective first phase only (see foregoing) or a central disorder with defects in both the first and last phases. One usually finds a falling tendency with poorer equilibrium reactions to the side on which a dissociated vestibular reaction in respect of the extremity reactions has been demonstrated. The falling tendency is usually directed towards this side,

Fig. 3. Patient 56. Organic central vestibular lesion. Tipping through longitudinal axis gives type I pathologic reaction. Both initial and final phases are defective to both sides.

The camera took 16 pictures a second. The tipping was maximal at $^{6}/_{16}$ seconds. The following pictures show the reaction in the final phase at $^{9}/_{16}$ and $^{12}/_{16}$ seconds.

but there are exceptions. Most of the post-traumatic vertigo cases with pathological tipping reactions belong to this group. The defensive reaction is good and the patient stands steadily when tipped to one side, but to the other side his extremity reactions are somewhat delayed, insufficient for the preservation of balance and, in the final phase, so poor, late, and insufficiently coordinated, uncertain and groping that it is difficult for him to adjust himself

to the new position. The stabilisation in the final phase takes a long time and balance is so poor that he stands and sways and falls to one side or the other, usually to the lowered side of the table. If one stops the table movement sharply the patient falls early in the final phase, often towards the raised side of the table, and it is impossible to study closely the nature of the reaction. In these cases the reaction is very typical and remains constant during repeated tests. In addition it must correspond with certain induced vestibular reactions. Restitution can occur and the tipping reaction then runs almost parallel with the improvement in the other vestibular extremity reactions. Sometimes there are exceptions, however, and one obtains the impression that examination on a tipping table is a more sensitive method of testing vestibular extremity reactions than ordinary vestibular stimulation. One may perhaps reach the threshold value for the pathological reactions more easily by examination on a tipping table than with caloric and rotatory tests, or it may be that the former method is more physiological. Figure 4 shows a series of reactions of this kind.

These patients usually react well to transversal table movements but the defence is often rather late and stiff. Ataxia is not so pronounced as in tipping to the sides and the nature of the reaction is often very difficult to judge. Transversal tipping is therefore used chiefly to complement longitudinal. The nature of the reaction is not typical in the same way as the latter and is also more complicated. Nor does it correspond with the rest of the vestibular reactions in the same way as after longitudinal tipping. Transversal tipping does not possess therefore the same practical value for the testing of cases of vertigo after head injuries. — If the patient is placed diagonally upon the table, and the table is tipped to one side just as in longitudinal tipping, then one obtains approximately the same reaction as in the latter. We have not found any possibility of arriving at a differentiated vestibular diagnosis beyond that yielded by ordinary tipping.

III. This type is dominated by the pathological alterations in the final phase. There is a rapid, lively defensive reaction which goes too far and is out of proportion to the disturbance of equilibrium on the table. It may also be somewhat delayed so that the patient first slips a short distance towards the tipped side after which there is a rapid hypermetric reaction which frequently gives him a tendency to fall over to the other side — the raised

Fig. 4. Patient 20. Organic central vestibular lesion. Tipping through longitudinal axis gives type II pathologic reaction to left. The initial and final phases are defective to the left. The reactions to the right are normal.

The camera took 16 pictures a second. The tipping was maximal at $^{6}/_{16}$ seconds. The following pictures show the reaction in the final phase at $^{9}/_{16}$ and $^{12}/_{16}$ seconds.

side of the table. The first phase is thus either too violent for the patient's ability to reach stability in his new position, or else it occurs so late that it seems to overwhelm the final reaction and the result is a hypermetric, violent, jerky and uncertain reaction which cannot preserve the patient's balance. The final phase is considerably prolonged before the patient arrives at a position of stability. If there is any falling tendency then it is frequently towards the raised side of the table. The ataxia is responsible for

the extremity reactions going too far and causing the patient to fall upwards. There is no definite rule, however, and the falling tendency as such is of no decisive diagnostic importance. Often one can demonstrate other signs of cerebellar disorder on the same side as the pathological tipping reaction, and this can be taken to support the objectivity of the test. — Transversal movements of the table also bring out atactic reactions, but they are often less clearly pronounced. — On the whole one can say that this type is marked by experimentally induced cerebellar ataxia which is shown up very clearly even in cases which clinically and neurologically yield no certain signs of cerebellar injury. One can always demonstrate pathological extremity reactions to ordinary vestibular stimulation at the same time — a dissociated series of vestibular reactions in respect of extremity reactions — and the tipping reaction as such can be used to verify them. The tipping reaction is more sensitive, however, yields clearer responses and is an easier method of examination. It does not of course exclude other tests. The reaction is illustrated in figure 5.

IV. In contrast to the other types, this one displays stiff, rigid reactions. The patient stands almost inanimate on the table and tumbles over to the lowered side of the table if the tipping is carried out completely. He stands with his arms and legs tense and fixed. There is usually a defensive reaction but the first phase is delayed and poor. The final phase provides poor stabilising movements which are stiff, small, slow and delayed. Adjustment to the new position takes very much longer and the most characteristic feature of this type lies in the final phase. The typical reaction both to longitudinal and transversal tipping, particularly the latter, is seen there. The first phase appears better or earlier in longitudinal tipping — at any rate one sees it more clearly in that movement. The head and extremities are held stiffly in a fixed position and do not follow the movement. The patients keep their balance better during slow movements and with the help of vision they react normally.

This form of reaction is seen sometimes where there are extensive head injuries, but also in old patients with cerebral atrophy or sclerosis without any pronounced local, central traumatic alterations. Corresponding pathological reactions in the form of dissociated vestibular extremity reactions are also manifested in cases belonging to this group. They do not possess any characteristic feature in themselves, however, and so this manner of react-

Fig. 5. Patient 10. Organic central vestibular lesion. Tipping through the longitudinal axis gives type III pathologic reaction to both sides. The initial phase is relatively good, at least to the right. The final phase is defective, with hypermetric movements and delayed adaptation on both sides.

The camera took 16 pictures a second. The tipping was maximal at $^{6}/_{16}$ seconds. The pictures in the last column show the reactions in the final phase at $^{9}/_{16}$, $^{12}/_{16}$ and $^{15}/_{16}$ seconds after tipping to the left.

The pictures after those displaying tipping to the right show the reactions when the table was slowly replaced to the original position. They were taken $^{12}/_{16}$, $^{18}/_{16}$ and $^{21}/_{16}$ seconds after the tipping was started.

Not until after about two seconds was any stable balance obtained in the final phase.

ing on the tipping table is surprising. The reactions to ordinary vestibular tests are rather slow, however, and sluggish, as the other somatic and psychic reflexes and reactions, and this may be

connected with and explain this particular form of reaction on the tipping table. There is no illustration to clarify this type.

Quite frequently, however, one comes across patients who behave in a similar manner in the beginning of a tipping table examination. They are usually elderly patients with normal vestibular reactions whose joints are more or less stiff and who become tense during the examination. They soon relax as the examination proceeds, however, and can learn to react normally, which is not of course the case where the reaction is pathological.

In this group, a diagonal position on the table causes no change in the reactions.

The position of the head does not seem to play any rôle in the extremity movements during tipping even in pathological cases. All the cases have been examined both with the head placed so that the horisontal semicircular canals are parallel to the horisontal plane and so that they are perpendicular to it, but no difference could be found in the reactions. This accords with the examination of normal cases previously mentioned on page 50 and with RADEMAKER's (71, p. 128) experience.

With regard to the movements of the head during tipping, pathological cases usually display the same movements — more or less pronounced — as normal cases. It has not been possible to demonstrate any general correlation between the various pathological extremity reactions and the movements of the head. Only in type IV can one discern a correspondence between the stiff, rigid extremity reactions and the stiff, fixed position of the head, but even then not always. In the other types, the head is twisted or bent slightly during longitudinal tipping towards the raised side of the table in the first phase, but returns to the central position during the final phase. If there is any falling tendency, the head is usually held in the central position. There are exceptions, particularly where there is recent labyrinthine injury with a reaction corresponding to type I (and II). In cases of bilateral labyrinthine destruction the head is usually held in the central position and does not react with any particular movement during the various tippings. Usually the patient stands and sways his head from side to side or else lowers it as far as possible. The same applies to cases of unilateral peripheral loss of the vestibular functions in its earliest stage, but as soon as compensation begins, the situation changes. In cases of central vestibular injury one can

usually see that there are clear head reactions even if they are not as precise and expressive as in normal cases.

The patients have also been examined in a sitting position and on the whole their reactions more or less correspond with those obtained in the hands and knees position when the cases are such as display a clear and pronounced falling tendency in the first phase. The correspondence is therefore greatest in types I and II and applies mainly to peripheral vestibular injury. Conditions are different where there are central vestibular changes. The vestibular tracts and pathways for the equilibrium of the trunk are separated from those which serve the extremity reactions (for instance in the arms) during tipping. One often finds therefore that a patient with a central vestibular injury who reacts in a pronounced pathological manner on hands and knees and whose other vestibular reactions are in correspondence therewith, reacts normally in a sitting position. Only in cases with more pronounced ataxia does one find a similar reaction in the different tipping positions, but in mild cases with small changes tipping in a sitting position is not so profitable from a diagnostic point of view as in a hands and knees position. The most important thing is, however, that examination in this position does not provide a check on the reactions seen in the other vestibular tests. One cannot verify the pathological reactions to tipping with the aid of dissociated vestibular reactions in regard to extremity responses as it is possible to do in the hands-knees position. This is the most fundamental factor for the value and usefulness of tipping as a method of examination. It seems more physiological to examine patients in a sitting position but the factors to be taken into account in judging the reaction are so numerous and so complicated that it is difficult to decide what is and what is not pathological in relation to purely vestibular equilibrium functions. Since the rest of the vestibular tests provide no check on the reactions, one has no means of evaluating one's discoveries. The result of all this is that I have not been able to use this method of examination by itself nor ascribe any decisive importance to it. It has been conducted as a matter of routine, however, just to see if it is of any diagnostic value. Examination in other positions, as Zador (101, 102) mentions in his works, seems for the same reasons to give equally small results in cases of head injury. Experiments have been made and they have always confirmed this assumption. The object of this work has also primarily been to

Tabular Survey

Approximate interval between injury and examination	Spontan. signs: Ataxia tests abnorm.	Spontan. signs: Pointing tests abnorm.	Spontan. signs: Romberg abnorm.	Spontan. signs: Gait abnormal	Spontan. signs: Spontan. nyst.	Positional nyst.: Type I (Nylén)	Positional nyst.: Type II (Nylén)	Positional nyst.: Only rotat., vertic. nyst. in hang. pos.	Caloric stimulation: Patholog. react. of peripheral type	Caloric stimulation: Patholog. response to optomotor stimulat.	Caloric stimulation: Patholog. nyst. response	Caloric stimulation: Vestib. tonus difference	Caloric stimulation: Dissociat. vestib. react.	Caloric stimulation: Eye deviation	Caloric stimulation: Normal react.	Rotatory stimulation: Patholog. nyst. response	Rotatory stimulation: Vestib. tonus difference	Rotatory stimulation: Dissociat. vestib. react.	Rotatory stimulation: Eye deviation	Rotatory stimulation: Normal react.	Tipping: About same fall. tendency to both s.	Tipping: Fall. tendency mainly to one side	Tipping: Hypermetric or similar react.	Tipping: Stiff, rigid or similar react.	Tipping: "Psychogenic" reaction	Tipping: Normal reaction
1 month																										+
				+	+			+											+			+				+
			+								+															+
1½ months																+										+
2 »																+										+
		+		+														+				+				
4 »	+							+																		
		+	+																							+
5 »				+		+													+			+				
			+				+																			
6 »		+						+								+						+				
				+																						
								+			not examined															+
7 »																+									+	
		+		+			+																			
10 »			+							L																
1 year																+									+	
			+																							
										R																

of Cases.

| Hearing | | |

Tabular Survey

Approximate interval between injury and examination	Induced Symptoms and Reactions: Tipping: Normal reaction	"Psychogenic" reaction	Stiff, rigid or similar react.	Hypermetric or similar react.	Fall. tendency mainly to one side	About same fall. tendency to both s.	Rotatory stimulation: Normal react.	Eye deviation	Dissociat. vestib. react.	Vestib. tonus difference	Pathолog. nyst. response	Caloric stimulation: Normal react.	Eye deviation	Dissociat. vestib. react.	Vestib. tonus difference	Pathолog. nyst. response	Pathолog. react. of peripheral type	Positional nyst.: Pathолog. response to optomotor stimulat.	Only rotat., vertic. nyst. in hang. pos.	Type II (Nylén)	Type I (Nylén)	Spontan. nyst.	Spontan. signs: Gait abnormal	Romberg abnorm.	Pointing tests abnorm.	Ataxia tests abnorm.
1½ years	+				+												R									
	+																R									
					+																					
2 »																										
2½ »																										
3 »																										
4½ »																										
5 »																										
6 »																										
7½ »																	R									
8 »																										
9 »																										

of Cases (continued).

Tipping reaction				Hearing						
First phase defective	Final phase defective	Peripheral defective hearing	Combined defective hearing	Neurogenic defective hearing	Deafness	Cases with norm. hearing & cases of defect. hearing not definitely of traumat. origin.	No. of patient	Age of patient	Conclusions and Comments
−	+	. .	. .	. .	R	L	36	37	Centr. vestib. dis. & peripheral incompensated dis. Same status after 6 months
+	+	. .	. .	. .	. .	+	37	35	Centr. vestib. dis.
++	+	. .	. .	. .	. .	+	38	40	» » »
. . . . .	. . . . .	. .	. .	. .	. .	+	39	55	» » »
+	+	. .	L	. .	R	. .	40	49	» » » & peripheral incompensated dis.
+	+	. .	+	. .	. .	+	41	50	» » »
. . . . .	. . . . .	. .	+	. .	. .	. .	42	50	"Vascular orig." Same stat. after 3 years
. . . . .	. . . . .	. .	. .	. .	. .	−	43	28	Centr. vestib. dis.
+	+	. .	. .	. .	. .	+	44	32	» » » Same stat. after 2 years
. . . . .	. . . . .	. .	+	. .	. .	. .	45	31	» » »
. . . . .	. . . . .	. .	. .	. .	. .	+	46	31	» » » Norm., Healthy after 2 months
++	+	. .	. .	. .	. .	+	47	24	» » »
. . . . .	. . . . .	. .	. .	. .	. .	+	48	19	» » »
+	+	. .	. .	. .	. .	+	49	19	» » »
. . . . .	. . . . .	. .	. .	. .	. .	+	50	45	» » »
+	+	. .	. .	. .	. .	+	51	26	» » »
+	+	. .	. .	+	. .	. .	52	43	» » »
. . . . .	. . . . .	. .	. .	. .	. .	+	53	28	» » »
. . . . .	. . . . .	. .	. .	. .	. .	+	54	41	» » »
+	+	. .	. .	. .	. .	+	55	27	» » »
+(+)	+	. .	. .	. .	. .	+	56	35	» » » Norm., Healthy after 1 year
+	++	. .	. .	. .	. .	+	57	41	» » »
. . . . .	. . . . .	. .	. .	. .	. .	+	58	42	» » »
. . . . .	. . . . .	. .	. .	. .	. .	+	59	44	» » »
+	++	. .	+	. .	. .	+	60	52	» » »
+	+	. .	+	. .	. .	+	61	52	» » »
. . . . .	. . . . .	. .	. .	. .	. .	+	62	47	» » »
+	+	. .	R	. .	L	. .	63	53	» » »
+	++	. .	. .	. .	. .	+	64	37	» » »
++	+	. .	+	. .	. .	. .	65	31	» » »
+	+	. .	. .	. .	R	L	66	26	» » » & peripheral incompensated dis.
++	+	. .	. .	R	L?	. .	67	52	» » »
+	++	. .	. .	. .	. .	+	68	61	» » »
. . . . .	. . . . .	. .	. .	. .	. .	+	69	60	"Psychogenic"

try to demonstrate a connection or correspondence between the tipping reaction in its usual form (longitudinal tipping) and the rest of the vestibular reactions in order to obtain some idea of its nature and value from a practical diagnostic point of view in otoneurological examination of cases of vertigo after head injury.

V. Patients with vertigo of neurotic, "psychogenic", origin exhibit normal reactions on the table with prompt and rapid defensive movements during fast tipping, but stand badly during slow tipping and fall more or less dramatically, usually in the direction of the lowered side of the table. Rapid tipping which takes the patient by surprise causes a lightning reaction and good adjustment in the final phase. Examination on a tipping table is therefore a suitable method in some cases to disclose exaggeration and simulation during the vestibular examination. This applies particularly to cases with striking spontaneous symptoms which react normally to induced vestibular tests and whose somatic nervous status is normal. They are very well able to manage the fastest tipping movements as long as they are quick and unexpected. The reaction is very typical with great swinging movements but nevertheless well adjusted for the maintenance of balance and with a well coordinated equilibristic first phase and a lightning sure adjustment in the final phase. One comes across this form of reaction now and again in head injury cases, due in part presumably to the fact that the patients seek in this way more or less deliberately to further their own interests. Within certain limits they can be definitely exposed by a tipping examination in conjunction with other vestibular tests.

E. Survey and Discussion of Cases Examined. Results.

If one now applies the method of examination previously described and judges the reactions observed on the principles already given, it will be possible to decide what connection and correspondence there is between some of the results of the various tests of vestibular function. For this purpose, the cases have been recorded on the foregoing chart which covers spontaneous signs, induced signs and reactions and hearing.

In this chart the spontaneous signs are recorded with a plus sign as also the induced signs and reactions demonstrated at the otoneurological examination. If the results of caloric and

rotatory stimulation and of the tipping table examination are normal, this has been denoted in the same way. In analysing positional nystagmus, NYLÉN's (65) grouping has been used in type I and II but instead of transitional nystagmus, there is a column for vertical nystagmus only in a supine position with the head hanging. This vertical nystagmus dominated, even if in some cases there was a rotatory factor in the nystagmus. There has been no change-over in this type of nystagmus and it has always been constant during different tests.

The nystagmus has been recorded in the same way as the previous results.

Special columns have been provided for the dissociated vestibular reactions, including pathological extremity reactions, to caloric and rotatory stimulation. These have included cases where deviation and pointing for a given stimulation have not been obtained or where the reaction has been otherwise pathological as previously mentioned. There must be correspondence between the results of caloric and rotatory stimulation, if this is functionally possible, for them to be regarded as definitely pathological. Examination of a series like the present one shows that this is usually the case.

Vestibular tonus difference has also been included in the columns which show the reaction to rotatory stimulation, because sometimes there was a nystagmus predominance in one direction which corresponded with the direction of the nystagmus obtained by caloric stimulation. It is obvious that one cannot with rotatory stimulation prove anything in this respect except a nystagmus increase in one direction or the other, and vestibular tonus difference of central origin cannot be demonstrated by rotatory stimulation for its typical feature is linked solely to caloric stimulation. It has been included, however, in order to serve as a check on the result of caloric stimulation. One usually finds an increased nystagmus tendency after rotatory stimulation and this nystagmus tendency possesses the same "Nystagmusbereitschaft" as after caloric tests. Even if the difference in duration is not particularly pronounced, there is increased frequency and intensity.

The reaction on the tipping table has been given in accordance with the five type groups mentioned on page 65. This type grouping is not ideal, of course, as far as the first four are concerned, for it does not take into consideration the essential features and value of the method. It must therefore be supplemented with a

column showing the nature of the reactions in the first and last phase during rapid tipping, which together with the attached details of the tipping provides a better picture of the patient's equilibrium disorder in a diagnostic respect and perhaps to some extent also in a topical respect. The nature of the reaction has been given in the usual way and the dominating phase has been shown by an increased number of plus signs.

With regard to the state of hearing in these cases of head injury it has usually not been possible to establish a definite connection between the hearing defect and the accident, except in cases suffering from deafness which further examination has shown to be almost certainly due to the trauma in question. The patient's profession, age, otoscopic changes, etc., have in most cases provided sufficient explanation of the hearing status, and the hearing defects have been judged with reference both to these considerations and to the nature and gravity of the accident (see p. 92).

The cases have been arranged chronologically on the chart on the basis of the approximate interval between the accident and the otoneurological examination. — In some cases there has been a later examination; the vestibular reactions then are given in relation to what was previously demonstrated. The last column also contains a brief comprehensive diagnosis and any remarks which may be of interest. — The approximate age of the patient at the first otoneurological examination is given in a special column.

1. The Classification of the Cases in Groups.

The chart shows all the cases which displayed a pathological vestibular reaction and the cases whose vestibular status was normal but which exhibited a positional nystagmus which was constant and combined with vertigo, although the latter was not linked with the nystagmus. Dizziness identical with spontaneous or positional vertigo of this kind did not develop when nystagmus with the same direction as that of the positional nystagmus was induced. Thus the vertigo is not in this case linked with a factor or partial function of vestibular origin but depends only upon the position. Apart from positional nystagmus nothing pathological could be demonstrated in these cases in the otoneurological tests. These cases have been placed in a special category and called "General vascular brain damage" or "Vertigo of vascular origin". In addition the cases were included which dis-

played a tipping reaction of psychogenic nature with a normal otoneurological condition and in which the neurological record did not point to any organic disorder as the reason for the reaction. The cases are classified in the same way as on page 27, except that the fourth group is excluded.

1. cases with certain organic vestibular disorder,

2. cases with signs of intracranial disorder, without any sign of vestibular disorder;

3. cases with a normal otoneurological and somatic nerve status but with a tipping reaction of psychogenic nature.

The fourth group is given in the former table on page 27 and covers cases which reacted perfectly normally to otoneurological examination and in which it was consequently impossible from our point of view to demonstrate anything pathological in the eighth cranial nerve and its connections or otherwise any signs of organic intracranial disorder. Neither did their neurological record provide any confirmation of symptoms or organic injury of such a kind that one could expect for certain a pathological series of reactions to otoneurological tests. The anamnesis in respect of vertigo was not as a rule such as to give the examiner any hope of making pathological discoveries.

In these cases the tipping reaction was always normal both to slow and rapid movements of the table and the vestibular partial functions reacted normally to caloric and rotatory testing. Spontaneous symptoms occurred quite frequently but it was not possible to confirm them by vestibular stimulation and I could not therefore ascribe any major importance to them. I have consistently judged the cases only on the basis of objective vestibular signs, i.e. on induced symptoms and reactions, and this has no doubt considerably reduced the number of cases in groups 1 and 2 and increased the number in the last group correspondingly. In a series such as this one often comes across cases where the neurologist cannot prove the existence of anything organically pathological but where the patient complains of dizziness. The physician who refers the case to us wants to know whether any organic vestibular defect can be demonstrated which explains the patient's vertigo, whether any sign of intracranial damage can be proved, or whether the vertigo is of psychogenic origin. If one can produce the patient's spontaneous dizziness in otoneurological examination and show that it is strictly linked with a factor of vestibular origin or if one can demonstrate the existence

of a pathological reaction to vestibular stimulation of such a kind that the patient's equilibrium disorder can be objectively explained as an organic disorder, then one can reply in the affirmative. This applies also to the discovery of signs of intracranial disorder of other kinds and not connected with purely experimental vestibular reactions. If, however, one cannot find anything pathological in the otoneurological condition, this does not necessarily mean that the patient's vertigo has no organic foundation. He may very well suffer from dizziness without our being able to explain it on organic vestibular lines with the methods of examination at our disposal. If, on the other hand, we can demonstrate the existence of an organic vestibular injury, it is not always certain that the dizziness is due to it or to it alone. Our knowledge of the anatomical and psychological background of dizziness is very small and we deal mainly with hypotheses. From an otoneurological point of view dizziness is attacked here as a problem of balance since it is one of the many symptoms of a defective equilibrating sense in which vestibular, stato-kinetic functions play the main rôle, in association primarily with visual functions and the deep and cutaneous sensibilities. If we can with the methods now at our disposal find an organic pathological reaction in these equilibrating functions which confirms the patient's complaint of dizziness and by which it can be experimentally induced and explained, then we shall have made considerable advance in our efforts to judge the complaint objectively. It is not often, however, that one can obtain a firm objective grip on the patient's sensation of dizziness, as is well illustrated by the fact that this last group was found to react absolutely normally from an otoneurological point of view without displaying any sign of intracranial disorder although there existed more or less pronounced dizziness. The result of all this is that I have never made any pronouncement in my reports as to whether or not the vertigo rests on an organic foundation or not, but have merely sought to decide whether any of our present tests reveal an organic pathological reaction. My opinions have therefore always been formulated with this consideration in mind. — Nonetheless, it has frequently been possible to verify the symptoms of vertigo objectively, particularly in the cases where it corresponds with the otoneurological observations. I have in mind particularly vertigo linked with nystagmus, either induced by position or by special changes of position, and the vertigo which occurs in cases of ves-

tibular tonus difference of central origin and in cases of dissociated vestibular reaction with pathological extremity reactions. The dizziness is then often typical and so individual that the patient can scarcely be suspected of inventing it. In dissociated reactions of this kind, pure equilibrating disorders overshadow the actual sensation of vertigo, for it comes comparatively late. Both here and in cases of vestibular tonus difference the vertigo disappears at the same time as the otoneurological reactions return to normal and there is, therefore, in all probability a connection between these special vestibular reactions and the vertigo. If the dizziness recurs, which is not unusual in a case of vestibular tonus difference then one can also demonstrate a typical series of reactions to otoneurological testing. The same applies in part to the vertigo linked with optokinetic troubles and which can be verified experimentally.

As a rule, the descriptions of the vertigo symptoms in the cases classed in this last group were not of such a nature or quality as to suggest that the otoneurological examination would yield any pathological signs. The symptoms were not of the kind which one usually encounters in cases of post-traumatic injury to the brain stem, but there were exceptions..

The first three groups are, therefore, of greater interest to us since it has been possible in these cases to demonstrate a pathological reaction in one or other respect during the otoneurological examination. It is on the basis of the results yielded by these examinations that an attempt will be made to prove that there exists a connection between a pathological tipping reaction and certain other abnormal otoneurological reactions. The examination on the tipping table was conducted in the same way in every case and one can see in the chart, firstly, how the patient reacted to rapid movements of the table and, secondly, his reactions on the same occasion to other tests such as caloric, rotatory, and optomotor stimulation and his nystagmus in various positions of the body and head and after changes of position. In addition the chart shows the spontaneous signs manifested at the same time and the condition of hearing. It has been constructed in this way so that it may be easy to overlook the results and to study them. It speaks for itself without any detailed descriptions and so I shall only deal closely with the factors which are of more interest for the purposes of this work.

Group 1.

The first group comprises cases where an organic vestibular disorder of some kind has been proved. These cases can be conveniently divided into two sections on the basis of the nature of the reactions.

1. *Vestibulo-ocular pathological reactions* characterized by only nystagmus changes on experimental vestibular stimulation. The reaction is of central vestibular origin and due to an organic change which affects the vestibular tracts and pathways for nystagmus. It is not possible to demonstrate any pathological extremity reactions but this does not, of course, prevent a dissociated reaction with pathological extremity movements occurring at the same time. In addition this section includes cases where eye deviation — "Bulbabweichung" — has been demonstrated. This is not a nystagmus reaction but a deviation of one eyeball in an abnormal manner when subjected to a certain stimulation. I regard this as a vestibulo-ocular reaction, even if it has been customary hitherto to class only pathological nystagmus in this category. It is practical to do so and no real objection can be raised to it. This group then comprises:

a. Pathological nystagmus response.
b. Vestibular tonus difference.
c. Eye deviation = "Bulbabweichung".

A pathological nystagmus response was the only abnormal sign in eight cases. In nine cases it occurred together with another pathological reaction, mostly a dissociated vestibular reaction. Thus it occurred in altogether seventeen cases.

Vestibular tonus difference is comparatively usual. In eleven cases the patient only displayed a typical reaction of this kind. The same sign was also observed in seven cases combined with a dissociated vestibular reaction, thus in eighteen cases altogether.

Eye deviation = "Bulbabweichung" has occurred in three cases. In one case it occurred together with a pathological nystagmus response and in the two other together with both a dissociated reaction and a pathological nystagmus response. In all the cases the ophthalmologist found normal conditions. The tipping reaction was normal in the first case and pathological in the two last. The value of eye deviation as an objective indication of central injury is not established for certain, but this reaction has been included because it has been found in cases of post-traumatic vertigo

(Stier and Beyer). It does not occur by itself, nor have I seen it by itself in other cases. For the time being it must perhaps be regarded as a secondary find of not certain importance.

Nor has optomotor stimulation yielded pathological reactions alone but always in conjunction with other reactions which were of decisive importance in judging the case. A pathological result can only be attributed definite importance in certain circumstances, and the examination therefore serves primarily as a check on or explanation of other reactions. In this series, a pathological result was found in two cases together with a pathological nystagmus response (nos. 1 and 43) and in three cases together with a pathological nystagmus response and a dissociated reaction (nos. 11, 36, 65). It has also occurred in four cases with purely dissociated reactions (nos. 21, 33, 38, 50) and in three cases with a vestibular tonus difference (nos. 15, 45, 53). Finally it has been found in case 10 together with all the other reactions mentioned. It is principally in cases with pure optokinetic symptoms that optomotor stimulation yields specific results, and in certain forms of vertigo linked with nystagmus of other kinds. — — — If one looks for the connection between the various types of optokinetic nystagmus — the cortical and the subcortical and the other forms of nystagmus which have been otoneurologically demonstrated, one often observes a correspondence between the subcortical form and that of purely vestibular origin. In this series, this connection was observed in the case of pathological nystagmus response, vestibular tonus difference and to some extent eye deviation also. Since I found subcortical optokinetic stimulation to be more profitable than cortical, most attention was given to the former.

In none of the cases with pure vestibulo-ocular reactions was examination on a tipping table able to demonstrate anything pathological, and the reaction was perfectly normal both to rapid and slow movements of the table and to all axes of movement. On the other hand, in cases where a dissociated vestibular reaction including pathological extremity reactions existed at the same time, the tipping reaction was pathological in one way or another to rapid tipping and normal to slow.

2. *Vestibulo-spinal pathological reactions* occurred most frequently in this series in the form of induced dissociated vestibular reactions with pathological deviation and pointing in relation to the nystagmus which occurred at the same time. They

occurred on stimulation of either the horisontal or vertical semicircular canals, and in every one of the cases the tipping reaction was pathological. Altogether there are 41 cases with this reaction, 27 of them only with dissociated reactions, 7 with a simultaneous vestibular tonus difference and 7 with a simultaneous pathological nystagmus response. Two of these cases yielded this reaction together with a vestibular tonus difference and a pathological nystagmus response. It has occurred in two cases (nos. 26 and 65) together with eye deviation and a pathological nystagmus response. In some of the cases with a pathological nystagmus response the vertigo was linked with this nystagmus. The same applies to some of the cases which display only a pathological nystagmus response. The dizziness is linked with a reaction of vestibular origin which in itself is pathologically changed.

In these forty-one cases, there are six with a peripheral vestibular injury with a dead labyrinth on one side. In these cases the nystagmus reaction was unusually well compensated for but the extremity reactions were defective. This condition cannot only depend upon a peripheral injury but must also be of central origin. Unilateral labyrinthectomy is usually compensated for in a few months, earlier in young than in older people. Six months after an accident, a 53 year old man did not exhibit compensation for his extremity reactions to ordinary vestibular stimulation, and his tipping reaction was pathological. A 37 year old man reacted in the same way ten months after injury but after a further year he becamed "healed", showed compensation and no symptoms. A 65 year old man reacted in the same way as the first-mentioned case a year after the accident, and two others one and a half years after their injury. One is 37 years old and the other 49. Finally there is a 26 year old man who as late as seven and a half years after the accident showed no compensation for his extremity reactions and whose tipping reaction was also pathological. Since the injury was in these cases a peripheral vestibular one and no compensation had developed after so long a time, it is highly probable that central vestibular changes are also responsible for the manner of reaction.

Thus the series contains no purely peripheral vestibular disorder — except perhaps the first of the above patients (no. 22). He displays vertical nystagmus in the supine position with head hanging which may be of pure "vascular origin", and independent of the trauma. The 65 year old man, (no. 33) reacts pathologically

to optokinetic stimulation *inter alia*, indicating a simultaneous central vestibular injury. In the fifth and last cases (nos. 40, 66) it is also possible that the injury is purely peripheral, but one thinks that compensation should have occurred after so long a time. The last patient is relatively young and compensation ought to have taken place in this and the other cases, if the injury were solely peripheral. — I have one case, not included in this chart, of a 26 year old soldier who had one tip of the petrous bone broken off in an accident causing a total loss of all labyrinthine functions on that side and slight commotio cerebri. In this case complete compensation with regard to the extremity reactions occurred after eight weeks. Subjectively he was by that time free of all symptoms and has since then not suffered from any vertigo or equilibrium disorders. It has not been possible to demonstrate objectively any central vestibular injury, and in this case the healing was very rapid.

Since the position of the injury in the above cases is not definitely known they have been described by themselves. Dissociated vestibular reactions, comprising pathological deviation and pointing and a pathological tipping reaction are common to all of them. In view of the latter, one can perhaps obtain further help in interpreting the injury. — The symptoms are both of peripheral and central origin, mostly the latter.

During optomotor tests one sometimes finds that dissociated vestibular reactions in respect of the extremity reactions can be demonstrated in inducing subcortical optokinetic nystagmus. The abnormal falling tendency seen after ordinary vestibular stimulation sometimes occurs here also, as well as abnormal deviation and pointing, and a certain correspondence between the subcortical reactions and those obtained by ordinary vestibular tests thus becomes more or less apparent. These reactions are not always constant, however, and one dare not therefore attribute great importance to them. They are rather mentioned as a circumstance worthy of note.

A survey of the occurrence and intermingling of vestibulo-ocular and vestibulo-spinal reactions is seen in table 3, which gives the case numbers of the patients in the columns for the different modes of reaction. If the patient has reacted in more than one way, his number is inserted in the columns for his different reactions. Nineteen displayed only vestibulo-ocular reactions and twenty-seven vestibulo-spinal reactions only. Altogether fourteen display

Table 3.

	Patholog. nyst. response Case Nos.	Vestibular tonus diff. Case Nos.	Eye deviation Case Nos.	Dissociated reactions Case Nos.
Patholog. nyst. response	1, 4, 5, 6, 23, 43, 59,62, 8			
Vestibular tonus diff.	*10*, *56*	3, 13, 15, 39, 45, 46, 48, 50, 53, 54, 58 = 11		
Eye deviation	(*4*), *26*, *65*			
Dissociated reactions	11, 16, *26*, 36, 44, 55, *65*	*10*, 14, 17, 20, 25, 47, *56*		2, 7, 9, 12, 19, 21, 22, 27, 30, 32, 33, 34, 37, 38, 40, 41, 49, 51, 52, 57, 60, 61, 63, 64, 66, 67, 68 = 27

more than one pathological reaction and of these, five display dissociated reactions at the same time as a pathological nystagmus response and five a vestibular tonus difference at the same time. Four of the fourteen displayed three pathological reactions. Two reacted with dissociated reactions + vestibular tonus difference + pathological nystagmus response. Two displayed dissociated reactions + pathological nystagmus response + eye deviation. See also page 86 and table 3.

Group 2.

The second group comprises the cases which showed signs of a central organic disorder although no signs were observed denoting injury to the eighth cranial nerve and its connections. The series includes two such cases, one of which is a 63 year old man (no. 8) who was examined two months after the accident. He had then positional nystagmus type I, and when a check was made four months later he had no symptoms and the status was normal. The second case was a 50 year old man (no. 42) with positional nystagmus vertically downwards only in a supine position with head hanging and without change of direction. The vertigo is in this case linked with position and not directly with the nystagmus. When the latter is induced in another bodily and head position no vertigo occurs corresponding to the spontaneous dizziness, and the

induced nystagmus is not pathologically changed. The vertigo is probably not therefore of vestibular origin. In the former case also, the vertigo was not accompanied by nystagmus. In the latter, a tipping reaction of psychogenic origin was also demonstrated and the condition one and a half years after the accident had not changed when checked one and three years later.

Group 3.

The third group in the series consists of the cases whose condition after the usual otoneurological examination was found to be perfectly normal, except that there was a positive Romberg and defective gait as spontaneous signs in some of the cases. No organic sign was demonstrated either here or under neurological examination. The tipping reaction was typical, with normal reactions to rapid tipping and specifically changed reactions to slow tipping. The tipping reaction was here of importance because it revealed their equilibrium defect as being psychogenic. It was the only test which gave a positive result in these cases. The psychological record showed that the reaction had been correctly interpreted, and the patient could be taught how to react. The rapid table movement showed him up and demonstrated that the equilibrium reactions corresponded to the normal otoneurological condition. It is in order to show that examination on a tipping table can be used to reveal simulation and exaggeration in some cases that this group was included. The reaction is very typical and has already been described on page 78. Altogether eight patients of ages varying from 29 to 60 years reacted in this way. One of them has positional nystagmus and has therefore also been classified in the preceding group. The patients have been examined repeatedly both neurologically and otoneurologically without any organic pathological signs being found. This does not necessarily mean — as I have pointed out before — that the patient's vertigo is not organic. By examining in this way one can give the physician who has sent in the case some positive information of value for judging and treating the case.

2. The Occurrence of Positional and Spontaneous Nystagmus in the Groups.

No spontaneous or positional nystagmus was demonstrated in group 3 with the exception of case 42, which also is assigned to

Table 4.

Manner of reaction	Positional Nystagmus			No positional nystagmus Case Nos.
	Type I Case Nos.	Type II Case Nos.	Only vertical in hanging position Case Nos.	
Patholog. nyst. response = a.	5, 43, 59	6, 62	23	1, 4
Vestib. tonus diff. = b	13, 48	3, 39, 45, 54, 58		15, 46, 50, 53
Dissociated react.	60, 61, 64	21, 66, 67	22, 34, 37, 38, 41	
Dissociated react. + a.	16	44, 55	11, 36	
Dissociated react. + b.		14, 17, 25, 47	20	
Dissociated react. + a + b.	10, 56			
»Of vascular orig.»	8		42	

group 2. There are 38 cases of positional nystagmus in groups 1 and 2 and by dividing them according to the various pathological vestibular signs one obtains the foregoing chart.

Spontaneous nystagmus has occurred in altogether seven cases, all belonging to group 1:

Vestibular tonus difference . . . . . Nos. 48 and 53.
Dissociated vestibular reaction . . . . . Nos. 21, 27, 52 and 63.
Dissociated vestibular reaction + patholog. nyst. response + vestib. tonus difference . . . . . No. 10.

One reason for compiling these tables is to see if there can be any connection between spontaneous or positional nystagmus and the nystagmus which on caloric stimulation appears as a vestibular tonus difference of central origin. If the direction of the spontaneous and positional nystagmus is the same as that of the latter then one is inclined to attribute to this the reason for "Nystagmusbereitschaft" according to Vogel. If one groups together all the cases in which a vestibular tonus difference was demonstrated then one obtains a table like the following. Apart from the cases given here there are however three cases in which neither spontaneous nor positional nystagmus was demonstrated

Table 5.

Patient No.	Spontan. nyst.	Positional nyst.	Vestib. tonus diff.	Result. of head movement test
3	0	type II r. ← positional vertigo	→ l.	
10	r. ← → l.	type I r. ← → l.	r. ←	
13	0	» I. r. ↶ ↷ l.	→ l.	
14	0	» II. r. ↶ ↓	r. ←	
17	0	» II. r. ←	r. ←	no influence
20	0	only vertical ↓	r. ←	
25	0	type II. ⇄ l.	↷ l.	
39	0	» II. → l.	→ l.	
45	0	» II. → l.	→ l.	
47	0	» II. r. ←	r. ←	
48	r. ← → l.	» I. ↓	→ l.	nyst. to l. of 2-nd degree
53	→ l.	0	→ l.	no influence
54	0	type II. ↷ l.	→ l.	
56	0	» I. r. ↶ ↷ l.	↷ l.	slight nyst. to l.
58	0	» II. r. ← ↓	→ l.	no influence

(nos. 15, 46, 50). Head movement tests yielded no pathological nystagmus in these cases.

In twelve cases, the direction of the spontaneous or positional nystagmus approximately coincided with that of the nystagmus which is a feature of the vestibular tonus difference. In cases No. 3, 20 and 58 the positional nystagmus has a quite different direction from that of the last-mentioned and in No. 48 its direction coincides with the predominating direction of the spontaneous nystagmus. Head movement tests in this case also demonstrate the existence of a latent nystagmus with this direction.

Thus we have altogether 6 cases in which the direction of the nystagmus is independent of any other nystagmus and twelve cases in which it more or less corresponds with the direction of the positional nystagmus. In the three cases with spontaneous nystagmus it corresponds, and the same applies to the cases with a latent nystagmus. The correspondence is greatest in these cases. A latent nystagmus could only be demonstrated in two cases.

One cannot draw any certain conclusions from the above because the series is too small. For safety's sake, one should not perhaps class as cases of vestibular tonus difference ones with the same nystagmus direction as a spontaneous or latent nystagmus and perhaps not either the ones with positional nystagmus of type II if the direction of the nystagmus is more or less the same. Literature on the subject gives no guidance, for the matter has not been explored (Van den Boorn, 22, Morsier and Barbey, 61). In examining this series no particular importance was attached to this reaction as regards diagnosis except in cases where no spontaneous, latent or positional nystagmus in the same direction could be demonstrated. In the other cases it was regarded as an additional observation which was made so often because the examination was conducted with such care. It was not possible or necessary to attribute any direct value to it in these cases since more certain pathological reactions were sufficient for an objective judgment. I have therefore mentioned this little matter chiefly for scientific consideration and investigation. — It is probably the case that a spontaneous nystagmus (latent) is made manifest as a result of the vestibular stimulation and is then diagnosed as a vestibular tonus difference.

Positional nystagmus has been demonstrated in 38 cases and is of the greatest importance as an objective indication of organic injury (Seiferth, 82). Nylén (68) points this out in examining post-traumatic conditions.

3. Regarding Hearing in the Groups.

It is very difficult to draw any conclusions from hearing changes in a series like this one for there are so many factors concerned. In most cases it is impossible to judge the part played by the trauma in the hearing defect. A hearing defect which corresponds to the otoscopic picture of traumatic origin can be more easily judged but it is usually impossible to decide whether the neurogenic and combined hearing defect with normal ear-drums has anything to do with the trauma or not. Age, heredity, earlier illnesses and working conditions can cause changes which make certain judgment in this respect impossible. For this reason some cases have been put in two or more columns in the table on page 74, both in the column which gives the quality of the hearing changes and in the last column: "Normal hearing + defective hearing of not

Table 6.

Manner of vestibular reaction	Reduced Hearing			No. of cases with norm. hearing + cases of defective hearing not of certain traumatic origin
	Combined	Neurogen.	Unilat. deaf.	
Vestibuloocular	No. 45			18
Vestibulospinal		No. 32, 52	No. 22, 27, 33, 40, 63, 66, 67	18
Vestibuloocular and spinal	No. 65	No. 10	No. 36	11
»Of vascular origin»	No. 42			1

definite traumatic origin." If one judges the hearing defects on the basis of these circumstances which are probably not connected with the trauma then one obtains a table like the foregoing.

Eleven cases with various hearing changes due to ear-drum defects, otitis, age, profession, etc. and where the patient has either stated that the hearing defect existed before the trauma or that hearing was not affected by the trauma, were placed in the last column. These were: No. 2; 67 years old with sclerotic ear-drums in which the hearing defect is probably senile. No. 4; with indented ear-drums, where the patient states that he has suffered from defective hearing for many years. No. 8 has for many years had large ear-drum defects after otitis. No. 11 is sixty years old and has had for a long time chronic otitis in the left ear and sclerotic ear-drum on rigth side. No. 16 is a 58 year old blaster with an old neurogenic hearing defect, the same on both sides. No. 33 is 65 years old with deafness in the right ear due to trauma and a hearing defect in the left ear since childhood which he does not consider to have been affected by the accident. No. 41 is a 50 year old machinist with an old hearing defect of the combined type, the same on both sides. No. 26 is a 37 year old man with defects after otitis on both sides and with a combined hearing defect, the same on both sides. No. 60 has defects after scarlatinal otitis and no. 61 has chronic otitis in one ear and an indented drum in the other. In nine of these cases was the hearing defect a combined one, and in two of them, nos. 2 and 16, it was neurogenic.

It appears from the table that hearing is generally not damaged to the same extent as the vestibular functions and this accords

with the fact that the series comprises almost wholly central injuries which affect the vestibular tracts and pathways in different directions. The exceptions to this are provided in part by cases with unilateral deafness, but in these cases central vestibular changes have also been found. The table is thus merely designed to show, with the foregoing reservations, the distribution of the hearing conditions in groups 1 and 2 and to show that hearing is comparatively unimpaired even where there are large and clear vestibular changes. The value of the hearing examination in a series like this lies principally in the fact that it serves as a check on the existence of a peripheral or central disorder in the eighth cranial nerve. It is, however, apart from the case of major changes, purely subjective and the result is usually difficult to judge in respect of the genesis. The testing of hearing has great defects — however it is carried out — and one has to rely wholly upon the patient's own testimony.

Skoog's (84) examinations show that in cases of head injuries in children the lasting injury is a hearing defect, but the vestibular functions are in the main satisfactory. In older persons, the state of hearing is difficult to judge, as appears also from Skoog's great work (83) and Schneider (81). According to Mygind (63), the hearing was impaired in nearly every one of his cases during the first period after the accident. The upper tone limit was lowered, but the bone conduction was usually not shortened. The reduction of the upper tone limit sometimes persisted, but nearly all patients gradually acquired "good hearing". — (Linthicum and Rand, 57).

4. The Relationship Between the Tipping Reaction and Other Signs.

A. Induced Vestibular Symptoms and Reactions.

In describing groups 1—3 I also touched upon the tipping table reactions. I will now try to illustrate the relationship between the tipping reaction and the other induced symptoms and reactions observed. If one looks to see with what otoneurological signs a pathological tipping reaction was associated, one obtains the following table.

It appears from this table that one obtains a pathological tipping reaction at the same time as a dissociated reaction embracing pathological extremity reactions — absence of or abnormal

Table 7.

		Otoneurologic observations	No. of cases	No. of cases with pathologic tipping reaction
Group 1.	Vestibuloocular	Patholog. nyst. response (a.)	8	0
		Vestib. tonus diff. (b.)	11	0
		Eye deviation (c.)	0	0
	Vestibulospinal	Dissociated vestib. reactions	27	27
	Vestibuloocular and spinal	Dissoc. vestib. react. + a.	5	5
		» » » + b.	5	5
		» » » + a + b.	2	2
		» » » + a + c	2	2
Group 2.	»Of vascular origin»	Positional nyst. + positional vertigo	2	0

deviation and pointing in the arms — to rotatory or caloric stimulation. In some cases the same correspondence exists between optomotor reactions of subcortical origin and a pathological tipping reaction. Pure vestibulo-ocular injuries yield a normal tipping reaction. A dissociated reaction so constituted that it only comprises nystagmus as a pathological function is not associated with a pathological tipping reaction. With regard to the peripheral + central disorders, no compensation occurred in these cases in respect of the extremity reactions, and there is a dissociated vestibular reaction involving defective extremity reactions. This category includes cases nos. 22, 27, 33, 36, 40, 66.

The cases in which both vestibulo-spinal and vestibulo-ocular changes were demonstrated, i.e. the cases previously given as combined, all displayed pathological tipping reactions. They totalled 14.

The cases in which positional nystagmus + positional vertigo was found without any other otoneurological pathological reactions, and which have therefore been classed in group 2, displayed a normal tipping reaction. The pathological reactions obtained during the examination gave rise to the assumption that there existed an organic central disorder which, however, did not influence the vestibular tracts and pathways. This cannot possibly be verified for certain with the means at present at our disposal.

The table thus shows primarily that a pathological tipping reaction coincided with dissociated vestibular reactions when they comprised extremity reactions to caloric and rotatory stimulation.

This relationship was constant and was substantiated by later examinations of cases not included in this series. Large numbers of persons suffering from producer-gas poisoning, which causes the same subjective and objective series of reactions as post-traumatic vertigo, have also all reacted in a corresponding manner (RÅDMARK, 80 a). Pure nervous cases have also at times displayed similar reactions. In some of the cases in the series, later examination has shown that both the dissociated reaction and the tipping reaction either remained unchanged or that the vestibular reactions were fully normal. One can observe the progress of compensation after a unilateral labyrinthine loss both with tipping and with ordinary vestibular examination at the same time and obtain results which correspond. Thus one can employ one method of examination as a check on the other.

The tipping table test conducted in the manner given here has therefore become a valuable addition to our methods of judging vestibular changes. A pathological tipping reaction + a dissociated vestibular reaction embracing pathological extremity reactions in respect of deviation and pointing in the arms provide a symptom complex which, in the cases of central vestibular injuries, can be called the *dissociated vestibular syndrome*. Examination on a tipping table provides a method of verifying objectively pathological deviation and pointing after caloric and rotatory stimulation. Hitherto we have not possessed such a method with the result that it has not been possible to judge the induced extremity reactions in the arms in the same manner as now. We have not been able to prove their organic origin and so they have frequently been regarded as of secondary importance. If one can now, as with this series, test the vestibular extremity reactions with two forms of stimulation — caloric and rotatory on the one hand and tipping on the other — then one can achieve greater objectivity because one reaction serves as a check on the accuracy of the other.

In other respects also, attempts have been in this series to judge the pathological vestibular symptoms on similar lines by trying to obtain the same reactions with different methods of stimulation. For if one can obtain the same subjective symptoms by more than one kind of objective stimulation, or if one can induce the same pathological reactions by different tests which are physiologically similar, then this is without doubt the surest method of disclosing an organic vestibular disorder. The one

Table 8.

Symptoms	1st Method of Examination	2nd Method of Examination
Positional vertigo or vertigo after changes of position	Examination in certain positions or after change in position causes positional nystagmus and corresponding vertigo	Experimentally produced corresponding nystagmus in other position shows that it is pathological, and in addition corresponding vertigo produced
Vertigo, uncertainty in quick movements, etc.	Dissociated vestibular reactions involving extremity reactions	Pathological tipping reaction
Vertigo on eye movements, optokinetics, etc.; uncertain feeling, etc. or vegetative symptoms	Pathological nystagmus response or vestibular tonus difference or eye deviation, etc. on caloric and rotatory stimulation plus corresponding subjective symptoms	Cortical or subcortical optomotor stimulation sometimes gives corresponding pathological nystagmus. sometimes eye deviation, etc. plus corresponding vertigo or vegetative symptoms

method of examination thus serves as a check on the objectivity and accuracy of the other. The method can be well illustrated by a couple of examples in table 8.

By conducting an examination on the lines of this example then one is in a better position to demonstrate pathological symptoms objectively. It is not always possible but one can try. The more developed is the method of examination the easier it is to demonstrate the abnormal vestibular reactions. The more ways we have of demonstrating a pathological reaction, the easier it should become. The tipping table examination gives us a further possibility in this respect with regard to the vestibulo-spinal changes, which makes it so valuable in the diagnosis of this type of lesions. It verifies and supplements pathological vestibulo-spinal reactions observed in the classical tests.

B. Spontaneous Signs.

We have now seen that there is a definite connection between the occurrence of a pathological tipping reaction and a dissociated vestibular reaction embracing the vestibulo-spinal connections, and that they comprise together a set of symptoms, a dissociated vestibular syndrome. But what is the relationship between the

Table 9.

	Spontan deviation, faulty pointing with arms to one side. (l. or r. side.)	Lowering of one arm (Moberg) (l. or r. arm.)	Manner of tipping reaction				
			Type I	Type II	Type III	Type IV	Normal
Pathолog. nyst. response (a.)	No. 59 l						No. 59
Vestibular tonus diff. (b.)	No. 13 l	No. 39 l.					No. 13, 39
Dissociated reactions	No. 7 l., 19 l., 27 l., 66 r.	No. 12 l., 30 l., 36 l., 64 r.	No. 12	No. 7 l., 19 l., 27 l., 30 l., 36 r.. 66 r.		No. 64 r.	
Dissociated reactions + a.		No. 55 l.		No. 55 r.			
Dissociated reactions + b.	No. 14 l., 25 l., 47 l.			No. 25 l., 47 l.	No. 14 bilat.		
Dissociated reactions + a + b.	No. 10 l.				No. 10 bilat.		

tipping reaction and the spontaneous symptoms, particularly deviation, past-pointing, ataxia, Romberg's sign and defective gait.

a. *Deviation and Past-Pointing in the Arms.*

Deviation and past-pointing have occurred spontaneously in one or both arms in altogether 16 cases. By entering them in the various columns for vestibular reactions and tipping reactions one obtains the foregoing table, where the side to which the tipping reaction was poorer is recorded.

The direction of the spontaneous deviation and past-pointing corresponds to some extent with the direction of the falling tendency during tipping. Exceptions are cases 10, 12, 14, 36 and 55. There is thus no direct correspondence in this respect. Past-pointing according to MOBERG (60) comprises a lowering of one arm with a slight medial deviation and the sign was comparatively weak in all the cases. No signs of paralysis appeared during clinical examination and the reflexes in the arms were normal.

It appears from the above that spontaneous deviation and past-pointing occurred also in cases with pure vestibulo-ocular signs

Table 10.

	Ataxia in:		Manner of tipping reaction				
	R. arm	L. arm	Type I	Type II	Type III	Type IV	Normal
Vestibular tonus diff.	No. 39						No. 39
Dissociated reactions	No. 12, 64, 68	No. 12, 51	No. 12	No. 51 l.	No. 68 r.	No. 64 r.	
Dissociated reactions + patholog. nyst. response		No. 36		No. 36 r.			

and a normal tipping reaction. In the 27 cases with pure dissociated reactions, spontaneous deviation and past-pointing occurred only 8 times, and in 5 out of 14 cases with mixed vestibulo-oculospinal lesions there were spontaneous signs. Thus in only 13 out of altogether 41 cases with a pathological tipping reaction was this spontaneous sign demonstrated, and one cannot suggest, therefore, that the two reactions correspond. For further data I would refer readers to the tabular survey on page 74.

b. *Ataxia in the Arms.*

Spontaneous ataxia in the arms was demonstrated in altogether six cases and has expressed itself as an insecurity in one or both arms during finger-nose and finger-finger pointing, during diadochokinesis tests and during the Stewart and Holmes test. The table foregoing, No. 10, shows what otoneurological reactions accompanied these spontaneous signs and the condition of the tipping reaction at the same time. The side to which the tipping reaction was poorer is recorded.

One can see in this table that this spontaneous ataxia occurred in one case with a pure vestibulo-ocular disorder and a normal tipping reaction. In the other five cases there are dissociated vestibular reactions and the tipping reaction is pathological. Not however, in more than a few of the large number of cases with dissociated reactions was it possible to demonstrate ataxia as a spontaneous sign. There is no correspondence with the tipping reaction but in the cases with the dissociated vestibular syn-

drome in which ataxia has been demonstrated the direction of the falling tendency corresponds with the side on which the ataxia in the arm is greatest — except in case no. 36. This is doubtless a mere coincidence. Spontaneous ataxia does not seem to have any effect upon the rest of the tipping reaction, for one would expect a tipping reaction of type III to be more frequent in ataxia cases.

Tests for these two spontaneous signs have been conducted with great care, but it is nevertheless not very frequently that they could be demonstrated although the induced reactions in many cases clearly suggested their existence. It is possible that I have been too critical in judging spontaneous signs, but I did not consider them certain unless they appeared constantly during repeated tests. Since the correspondence with the objective dissociated reactions was as small as in this series I did not either dare to ascribe any decisive importance to them. Only in a small number of the cases which displayed abnormal vestibulo-spinal reactions to caloric and rotatory stimulation did I observe these spontaneous signs in the upper extremities. In three cases with positive spontaneous signs, the examination did not reveal any induced pathological extremity reactions. — In a series such as this, in which the tendency to simulate and exaggerate is often very great and must therefore always be taken into account, spontaneous signs must not be ascribed any certain importance in a diagnostic respect unless there is direct correspondence with more objective vestibular signs. These remarks regarding the relationship between spontaneous signs and induced vestibulo-spinal reactions apply, of course, also to the pathological tipping reaction.

c. *Romberg's Sign.*

The two other spontaneous signs — Romberg's sign and defective gait — given in the tabular survey stood on about the same level with respect to their diagnostic value. They were usually very difficult to judge with certainty and the subjective, psychogenic factor in the set of reactions was often very strong. If one selects the cases where the Romberg test yielded a positive result in this series, one obtains the table below.

There is no direct correspondence between the details of an abnormal reaction to the Romberg test and of the tipping reaction, as the table shows. The direction of falling tendency during tipping has been given beside the patient's number.

Table 11.

	Romberg's test				Manner of tipping reaction					
	Falling tendency:									
	undecid.	backw.	to r.	to l.	Type I	Type II	Type III	Type IV	Type V	Normal
Vestib. tonus diff. (b.)		No. 3, 48								No. 3 48
Dissociated reactions	No. 21, 22, 63, 67	No. 40, 60, 68	No. 33, 41, 66,	No. 30, 34, 51, 64	No. 21, 22 r.	No. 30 l., 34 r., 40 r., 41 r., 51 l., 63 l., 66 r., 67 r.	No. 68	No. 33 r., 60, 64 r.		
Dissociated reactions + patholog. nyst. response (a.)	No. 55		No. 36			No. 36 r., 55 r.				
Dissociated reactions + b.	No. 17			No. 47		No. 17 r., 471				
Dissociated reactions + a + b.	No. 10						No. 10			
Dissociated reactions + a + eye deviation	No. 26, 65				No. 65 r.	No. 26 l.				
»Of vascular origin.»		No. 42							No. 42	
»Psychogenic.»	No. 29, 69	No. 28							No. 28, 29, 69	

In one way or another, the result of the Romberg test was regarded as positive in 27 cases. In about half of the cases where the dissociated vestibular syndrome was demonstrated, Romberg's sign was observed. Thus neither is there any direct correspondence between this spontaneous sign and the induced reactions. In 4 of the 8 cases with a tipping reaction of psychogenic type, the Romberg test was positive.

d. *Defective Gait.*

In altogether 32 of the cases in the three groups there was a deviation or other abnormality during walking tests with eyes

covered. Table 12 shows the relationship between these reactions, the result of otoneurological examination and the tipping reaction. In some cases one sees a certain correspondence between the direction of the deviation during walking tests and the direction of the falling tendency during tipping. In most cases the latter is not very pronounced but the side given is the one to which the patient made inferior subjective and objective defensive movements. As a rule there was no actual fall.

The foregoing remarks regarding the Romberg test apply also to the gait examination. There is usually no direct correspondence between this spontaneous sign and induced signs and reactions, but sometimes one does see a certain connection between a positive Romberg and a gait deviation to one side with a poorer tipping reaction to that side. This matter will be dealt with more closely below.

A positive Romberg and defective gait have occurred together in twenty-six cases, a positive Romberg alone in one case (no. 34) with injury to the vestibulo-spinal pathways. In four cases — nos. 2, 16, 20 and 44 — there was only a gait defect, the injury in case no. 2 being of the same type as in no. 34, while the others had injury both to the vestibulo-spinal and to the vestibulo-ocular pathways. In all these five cases the tipping reaction was pathological.

These remarks about the spontaneous signs show that one does not always obtain correspondence between them on the one hand and induced vestibular reactions on the other, and this applies to the tipping reaction also. Spontaneous ataxia, deviation or past-pointing does not always accompany a pathological tipping reaction. There are altogether 41 cases with a pathological tipping reaction in this series (the psychogenic cases not included) and if one adds up all the cases with the spontaneous signs given in the table one arrives at a total of 40. But of these patients, only 31 have a pathological tipping reaction. The others which reacted pathologically on the tipping table *did not* display *any* of the above spontaneous signs. Spontaneous signs were also found in five cases which displayed fully normal tipping reactions and in four whose reactions were psychogenic. In 2 cases, all four spontaneous signs occurred together and in 7, three of them. Of the rest, two signs have been found together in 17 of the cases. Of these 26 cases with more than one spontaneous sign, the tipping

Table 12.

	Defective gait.			Manner of tipping reaction					
	Swaying without definite deviation	Deviation: to r.	Deviation: to l.	Type I	Type II	Type III	Type IV	Type V	Normal
Vestibular tonus-difference (b.)	No. 48		No. 3						No. 3, 48
Dissociated reaction	No. 21, 22, 40, 41, 60, 63, 64, 67, 68	No. 2, 33, 66	No. 7, 30, 51	No. 21, 22 r.	No. 2 r., 30 l., 34 r., 40 r., 41 r., 51 l., 63 l., 66 r., 67 r.	No. 7, 68 r.	No. 33 r., 60, 64 r.		
Dissociated reaction + pathol. nyst. response (a.)	No. 16, 55	No. 36	No. 44	No. 16 r.	No. 36 r., 55 r.				
Dissociated reaction + b:	No. 17, 47		No. 20, 25		No. 17 r., 20 l., 25 l., 47 l.				
Dissociated reaction + a + b.	No. 10					No. 10			
Dissociated reaction + a + eye deviation	No. 65		No. 26	No. 65 r.	No. 26 l.				
»Of vascular origin.»	No. 42							No. 42	
»Psychogenic.»	No. 28, 29, 69							No. 28, 29, 69	

reaction was pathological in 23. The relationship between the tipping reaction and the occurrence of the various spontaneous signs can be seen in the preceding tables.

For a closer study of the relationship between the various reactions I would refer readers to the tabular survey on page 74, where the details can be examined from every possible point of view. It is obvious that in judging these questions the quality of the spontaneous signs is decisive. But the reactions which are regarded as pathological are, in the case of spontaneous signs, comparatively similar in this series. The prime object in this case is to study their relationship with the tipping reaction in order if possible to trace some correlation between them. Sometimes one finds such correlation and cannot say for certain why. Often the

spontaneous signs are very subjective with the result that their value in the examination is perhaps ignored and overlooked. Another factor which may perhaps explain the incongruence is that the organic changes are so small that they do not appear as spontaneous signs but become manifest only on experimental vestibular stimulation.

5. Regarding Different Pathological Reactions on the Tipping Table in Relation to Spontaneous Signs and to Induced Signs of Defect in the Vestibular Functions.

It appears from the foregoing that the pathological tipping reaction has expressed itself in various ways, and I have preliminarily divided it into four types.

a. *Tipping Reaction of Psychogenic Nature.*

Cases of vertigo in which no organic vestibular disorder could be demonstrated and in which the tipping reaction was perfectly normal during rapid movements of the table but in which the patient reacted in a particular manner to slow movements while neurological examination could not find any organic reason for the reactions, were classed in a fifth type (see page 78). This latter particular reaction to slow table movements was therefore classed as "psychogenic" and the examination on the tipping table made it possible to expose the patient. These cases have been included and their reactions described because they provide a further use for tipping table examinations. The reaction is very typical in many cases, but one must carefully look for every possible organic explanation before classing it as psychogenic. One merely informs the physician who has submitted the case that the patient reacts to examination in this way and it then rests with him to find the reason. There are eight cases in this series which react in this way. In seven cases no organic pathological disorder of any kind was found. One patient, no. 2, has positional nystagmus + positional vertigo. The otoneurological condition is perfectly normal and it is difficult to explain why he, and the other seven, react in this way on the tipping table since no organic change to explain it can be found.

The cases whose pathological reactions on the tipping table are of organic origin are divided as follows into four types:

Table 13.

Type I About same falling tendency to both sides	Type II Falling tendency more to one side	Type III Hypermetric or similar reactions	Type IV stiff, rigid or similar reactions	Type V »Psychogenic» reactions
No. 12, 16, 21, 22, 38, 56, 65, 67	No. 2, 7, 9, 17, 19, 20, 25, 26, 27, 30, 32, 34, 36, 37, 40, 41, 44, 47, 49, 51, 52, 55, 61, 63, 66	No. 10, 11, 14, 68	No. 33, 57, 60, 64	No. 18, 24, 28, 29, 31, 35, 42, 69

These types are not strictly separated from each other in respect of their reactions for the characteristic feature of each type is merely the dominating factor in the actual reaction. A falling tendency to one side is often accompanied by a pathological reaction to the other side also. In type III there is often a falling tendency to both sides, but there are other factors in the reaction which are clearer than this and so dominate the set of reactions that it acquires a typical appearance. The same applies to some extent to the last type. The division into these different types may therefore seem a little artificial but it has been done in order to facilitate the study of the tipping reaction on the one hand and its relation to the rest of the otoneurological condition on the other, in order to try to explain why the patient reacts in such and such a manner on the tipping table. Is there any correspondence between the special manner of tipping reaction and the individual features of the induced dissociated vestibular reaction? Is there perhaps any similar correspondence between the spontaneous signs and the tipping reaction? — Rademaker (71, p. 115) declares that he cannot lay down any definite rule based on the tipping reaction in cases of injury to the vestibular apparatus on one side or the other. Zador (102, p. 128 etc.) wonders if there exists any relationship between the direction of the pathological tipping reaction and the position of the injury; i.e. both the side on which it is situated and its central location. He gives no definite reply but describes a case with post-traumatic bleeding and lesion on the pons of the left side below the nucleus ruber, but vestibular injury on the right side. The patient had astasia, abasia and a right-sided hemiplegia and incomplete rigidity. When examined on the tipping table the patient fell when tipped to the left but displayed

defensive reactions to the right. In transversal movements he fell consistently to the left. In this case the direction of fall corresponds with the side of the central injury. This case is of no value as a proof and is very complicated both from the standpoint of symptoms and pathologic anatomy, even if a correlation exists between the location of the injury and the direction of falling tendency during tipping. He points out, however, that it is impossible to obtain any fixed relationship between the direction of the pathological tipping reaction and the side which is vestibularly injured. His investigation was conducted on a purely neurological basis and on a purely neurological clientèle, which is best illustrated by his attempts to prove a correspondence between the clinical form of ataxia and the tipping reaction. The present series is a very different one and the relationship between spontaneous signs, induced vestibular signs and reactions and the tipping reaction is to be illustrated from an otoneurological point of view. The forms of organic central injury in this series were less grave than in ZADOR's case.

If one examines the cases in the various type-groups and ascertains where in the otoneurological status the defect is mainly situated after caloric and rotatory stimulation, and observes the appearance of the defect and the spontaneous signs obtained at the same time, then one obtains tables such as the following. Since the pathological tipping reaction can be considered solely from a vestibular point of view to be linked with the occurrence of dissociated vestibular reactions which embrace extremity responses, the special features of the tipping reaction types should also be linked with the same reactions and thus to some degree correspond with the individual features of the latter. This applies if the defect involves a part of the vestibular tracts and pathways for the inducement of extremity reactions, i.e. from the vertical or horisontal semicircular canals, and also to the appearance of the induced extremity reactions.

b. *Type I.*

Eight cases reacted on the tipping table in a manner corresponding to type I and these cases have been entered in the table below.

In these cases there is a falling tendency to both sides or else in all axes of movement during the tipping table examination (see fig. 3). One finds sometimes, however, that the falling tendency is

Table 14.

Patient No.	Spontan. symptoms	Diss. react. on caloric stimulation of: (semicirc. canals)	Extent and direction of extremity reaction defect:	Diss. react. on rotatory stimulation of: (semicirc. canals)	Extent and direction of extremity reaction defect:
12	Ataxia: in both arms Pointing: l. arm deviate medially (Moberg)	vertical	absent, bilat., to both sides	vertical	absent, bilat., to both sides
16	0	»	»	»	»
21	Romberg: no decid. fall. tendency. Gait: no decid. deviation	»	»	»	»
22	»	not examined	—	»	»
38	0	vertical	absent, bilat., to both sides	»	»
56	0	»	»	»	»
65	Romberg: no decid. fall. tendency Gait: no decid. deviation	» + horisontal	weak, absent, bil., to both sides	»	weak, bilat., to both sides fr. horisont. absent, bilat., to both sides fr. vertical.
67	»	vertical	absent, bilat., to both sides	»	absent, bilat., to both sides

more pronounced to one side although the reaction is not for that reason normal to the other. Among the spontaneous signs one finds no decided falling tendency and no pronounced walking deviation to one side or the other. Ataxia occurred in one case and then in both arms. The dissociated vestibular reactions were usually connected with the pathways for the vertical semicircular canals on both sides. Case 65 displayed corresponding defective reactions also from the horisontal semicircular canals on caloric stimulation, but no reaction to rotatory stimulation of the vertical. The pathological extremity reactions induced by caloric and rotatory stimulation usually embraced both arms and mostly took the form of an absence of extremity reaction to both sides and in both arms or else resembled case 12, which has no definite reac-

tion on either side but in which the left arm also drops inferomedially as in the spontaneous test. The past-pointing in this case is of the type which MOBERG has described. No. 22 has deafness in one ear, a peripheral + central vestibular injury which is not compensated for in respect of the extremity reactions. The caloric stimulation was incompletely carried out in this case.

The common features of this type group are thus that the deviation and pointing after stimulation were usually absent or greatly reduced to both sides in both arms, mainly on stimulation of the vertical semicircular canals. The spontaneous signs comprised a positive Romberg and gait defect without any decided falling tendency. The tipping reaction is typical with poor defence to both sides or for all axes, particularly in the first phase.

c. *Type II.*

If one now compiles a corresponding table for the cases which display more or less pronounced falling tendency or poor equilibrium reactions to one side, but manage better or stand normally to the other, then it should be easier to see if there is a correspondence between the different reactions since they are more finely shaded in the case of the tipping reaction. Type group II in the series embraces the largest number of cases.

All one really sees from the following table is that one sometimes obtains a correspondence in direction between the falling tendency in the Romberg test and, to a more pronounced degree, in walking deviation, on the one hand and the tipping reaction on the other. Spontaneous past-pointing and deviation do not correspond so well, however, nor does ataxia in the arms. One cannot draw any conclusions from their relationship with the tipping reaction in the matter of falling tendency to one side or the other. Dissociated vestibular reactions with pathological extremity reactions occurred mainly on stimulation of both the horisontal and vertical semicircular canals in the same patient. There are only three cases in which this pathological reaction was linked solely to the vertical semicircular canals (nos. 9, 47 and 55). All three really constitute borderline cases between this and the preceding type.

One cannot demonstrate any certain connection between the dissociated reactions and the direction of the falling tendency in the tipping test. It is certainly a fact that the difference between the reactions to one side or the other is in most cases very small but that the falling tendency emphasises the defective extremity

Table 15.

Patient No.	Spontan. symptoms	Diss. react. on caloric stimulation of: (semicirc. canals)	Extent and direction of extremity reaction defect	Diss. react. on rotatory stimulation of: (semicirc. canals)	Extent and direction of extremity reaction defect	Manner of tipping reaction
2	Gait; deviat. to r.	vertical + horisont. on r. side	absent to r., bilat.	vertical + (horisont.)	greatly reduced. absent to r., bilat.	poorer to r.
7	Gait: deviat. to l. Point.: deviat. to l. in both arms	vertical + horisont.	absent, bilat., to r.	vertical + horisont.	absent, bilat., to r.	» » l.
9	0	vertical on l. side	absent, bilat., to both sides	vertical	absent, bilat., to both sides	» » »
17	Romberg: no decid. fall. tendency Gait: no decided deviat.	vertical + horisont.	weak, bilat., to both sides	vertical + horisont.	indistinct, uncertain bilat., to both sides	» » r.
19	Point.: deviat. to l. in both arms	vertical + horisont. (particular. r.)	greatly reduced. absent to r., bilat. absent from vertical r.	»	greatly reduced, absent, bilat., to both sides	» » l.
20	Gait: deviat. to l.	vertical + horisont.	no certain reactions, bilat., partly inverse from horisont.	»	absent to r. in r. arm, crossed with l. arm. (falling tendency to l.)	» » »
25	Point.: deviat. to l. in both arms. Gait: deviat. to l.	»	greatly reduced, bilat., to r.	»	absent, to r., bilat., from vertical, weak from horisont.	» » »
26	Romberg: no decided fall. tendency Gait: deviat. to l.	»	absent, bilat., to both sides	»	absent, bilat., to both sides	» » »
27	Point.: deviat. to l. in both arms	» (l. labyrinth dead)	reduced in r. arm, crossed to r. with l. arm	»	absent, bilat., to r. fr. vertical, weak fr. horisontal	» » »

Patient No.	Spontan. symptoms	Diss. react. on caloric stimulation of: (semicirc canals)	Extent and direction of extremity reaction defect	Diss. react. on rotatory stimulation of: (semicirc. canals)	Extent and direction of extremity reaction defect	Manner of tipping reaction
30	Point: lowers l. arm. Romberg: fall. tendency to l. Gait: deviat. to l.	vertical + horisont. on r. side	weak, bilat., to both sides	vertical + horisont.	greatly reduced, bilat., to l. exaggerated, bilat., to r.	poorer to l.
32	0	vertical + horisont.	weak, indistinct, bilat., particular. to l.	»	greatly reduced, indistinct, bilat., to both sides	» » r.
34	Romberg: fall. tendency backwards	»	weak, indistinct, bilat., to both sides	»	» and more reduced to r.	» » »
36	Ataxia in l. arm Point: lowers l. arm Romberg: fall. tendency to r. Gait: deviat. to r.	» (r. labyrinth dead)	no certain reactions, bilat., to both sides (fall. tendency to r.)	»	absent in l. arm to both sides	» » »
37	0	vertical + horisont.	absent, bilat., to r. side fr. vertical, greatly reduced, bilat., to both sides fr. horisont.	»	absent, bilat., to r. fr. vertic. weak, bilat., to r. fr. horisontal	» » »
40	Romberg: fall. tendency backwards Gait: no decid. deviat.	horisontal (vertical not examined) (r. labyrinth dead)	greatly reduced, bilat., to both sides	»	absent, greatly reduced, bilat., to both sides	» » »
41	Romberg: fall. tendency to r. Gait: no decid. deviat.	vertical + horisont.	»	»	greatly reduced, indistinct, bilat., to both sides	» » »
44	Gait: deviat. to l.	»	»	»	absent, bilat., to both sides fr. vertical, weak fr. horisontal	» » l.

Patient No.	Spontan symptoms	Diss. react. on caloric stimulation of: (semicirc. canals)	Extent and direction of extremity reaction defect	Diss. react. on rotatory stimulation of: (semicirc. canals)	Extent and direction of extremity reaction defect	Manner of tipping reaction
47	Point.: both arms deviat. to l. Romberg: fall. tendency to l. Gait: deviat. to l.	vertical	absent, bilat., to both sides	vertical	absent, bilat., to both sides	poorer to l. (about equally to both sides)
49	0	» + horisontal	greatly reduced, weak, indistinct, particular. in l. arm, to both sides	» + horisontal	greatly reduced, indistinct, bilat., particular. in l. arm, to both sides	poorer to l.
51	Ataxia in l. arm Romberg: fall. tendency to l. Gait: deviat. to l.	»	greatly reduced in l. arm and weak in r. arm, to both sides	»	greatly reduced in l. arm, weak in r., to both sides	» » »
52	0	horisontal (vertical not examined)	weak, bilat., to both sides	»	no definite reaction, bilat., to both sides	» » r.
55	Point.: lowers l. arm Romberg: no decid. fall. tendency Gait: no decid. deviat.	vertical, especially l.	weak, indistinct, bilat., to both sides	vertical	absent, no definite reaction, bilat., to both sides	» » »
61	0	vertical + horisontal on the l. side	weak indistinct, bilat., to both sides	vertical + horisont.	weak, bilat., to r. side, no definite reaction to l. side	» » »
63	Romberg: no decid. fall. tendency Gait: no decid. deviat.	vertical + horisontal (left side)	no definite reaction, bilat., to both sides	vertical + horisont.	no definite reaction, bilat., to both sides from vertical and to l. from horisontal	» » l.
66	Point.: both arms deviate to r. Romberg: fall tendency to r. Gait: deviat. to r.	vertical + horisont. (r. labyrinth dead)	absent, greatly reduced, bilat., to l.	»	absent, bilat., to l.	» » r.

reaction more clearly to one side. Often the patient says that he finds it more difficult to manage the tipping to one side than the other. He says that he cannot follow the movement so well. If he displays a positive Romberg or walking deviation to one side then one often finds that he stands somewhat less well when tipped to that side. It is seldom, however, that the patient falls, for the defensive reactions exist although they are poorer to one side. One gains the impression that a case with a positive Romberg and a walking deviation to one side finds it still more difficult to manage tipping to that side. One feels that a clear ataxia in one arm should affect the falling tendency in the tipping test, but this is not always the case. No. 36 has marked ataxia in the left arm which does not follow the direction of pointing on caloric and rotatory stimulation. In the tipping test, he says that he finds it more difficult to follow the table movements to the right than to the left. If one completes the table movement rapidly one sees that he shows a certain tendency to fall to the right. The Romberg and the walking tests also show a falling tendency and deviation to the right. It is difficult to decide what most influences this special manner of tipping reaction — the ataxia or the statokinetic equilibrium disorder. Probably it is the latter which plays the greatest rôle.

The direction of the defective extremity reaction when the patient is subjected to caloric and rotatory stimulation may correspond with the direction of the falling tendency in the tipping reaction. In five cases there is spontaneous deviation and past-pointing in the arms to one side and the defect is here largest to the other side; the falling tendency in the tipping reaction is here contrary to the direction of the most pronounced induced defective extremity reaction.

One cannot draw any definite distinct conclusions beyond what has already been said regarding correspondence with the Romberg and walking tests. A disorder causing a pathological reaction to the same side under these tests seems to increase any existing tendency to fall to this side during tipping.

The following characteristic features of this type can be mentioned: The tipping reaction is less well pronounced to one side (see fig. 4). The defective extremity reaction is usually induced by stimulation both of the horisontal and vertical semicircular canals. The direction of the induced defective extremity reaction does not always coincide with the direction of the falling tendency

during tipping. With regard to spontaneous symptoms, gait and standing tests are usually pathological to one side and a certain correspondence can be discerned between the direction of the deviation and falling tendency and the direction of the falling tendency during tipping.

d. *Type III.*

ZADOR (102, p. 146) declares that it is not possible to predict how cases of mild static and kinetic ataxia will react on the tipping table. If astasia and hypotonia occur at the same time as ataxia, however, the result is a pathological tipping reaction which can be divided into two types. The first reacts with hypermetric movements and a falling tendency contrary to the direction of the table movements, and the latter with a falling tendency in all directions. These observations of his were made in nervous cases.

In this series, static ataxia does not correspond definitely with the tipping reaction, but there is more correspondence when the ataxia is kinetic. This is quite natural since the kinetic equilibrium should make itself felt particularly during examination on a tipping table. The static form appears most clearly during the final phase of the reaction, and it can then, if it is defective, yield a reaction which corresponds to one of the types previously described by ZADOR. Cases which reacted with hypermetric or similar reactions have been classed with this third type of pathological tipping reactions, which comprises four cases. See table 16.

The defensive reaction to table movements is very rapid in these cases but is not balanced in a suitable way. It is too strong and often goes beyond the mark so that the patient tends to fall towards the raised side of the table (see fig. 5). The poor coordination and symmetry of the extremity movements, which prevents an efficient equilibrium reaction, is particularly apparent. One obtains large, swinging, unbalanced hypermetric extremity and body movements. The first case, No. 10, is a typical one of this kind. One does not find much in the neurological record: "Somatic: no particular features. Psychic: easily fatigued, good judgment, no exaggeration. Speech clearly cerebellar, no aphasia. Homonomous diplopia originating in paralysis of the left obliquus inferior muscle. Esophoria." One cannot demonstrate any ataxia in these case by the usual methods, but it comes out beautifully during examination on the tipping table. It is just in this respect that this case differs from ZADOR'S cases previously mentioned.

Table 16.

Patient No.	Spontan. symptom	Diss. react. on caloric stimulation of (semicirc. canals)	Extent and direction of extremity reaction defect	Diss. react. on rotatory stimulation of (semicirc. canals)	Extent and direction of extremity reaction defect
10	Point.: both arms deviate to l. Romberg: no decid. fall. tendency Gait: no decid. deviation	vertical + (horisont.)	greatly reduced. bilat., to both sides	vertical	greatly reduced, bilat., to both sides
11	0	vertical	absent, greatly reduced. bilat., to both sides	»	absent, greatly reduced, bilat., to both sides
14	Point: both arms deviate to l.	»	»	»	absent to r., weak, uncertain to l. in both arms
68	Ataxia: r. arm and r. leg. Romberg: fall. tendency backwards. Gait: no decid. deviation	»	indistinct, greatly reduced, sometimes crossed, bilat., to both sides	»	indistinct, greatly, reduced, sometimes crossed, bilat., to both sides

A characteristic feature of this group is the special tipping reaction with its pronounced ataxia, particularly in the final phase. The falling tendency may be to either side, for it is most dependent on the direction of the table movement. Spontaneous signs — Romberg and gait — display no decided falling tendency or deviation in any direction. In No. 10 and 14 there is a deviation in the arm to the left and No. 68 has ataxia in the right arm and leg. In the two former cases the reaction to tipping examination is more or less the same to both sides, and the spontaneous deviation does not therefore seem to make itself felt in this examination. In the latter case, on the other hand, one can discern an inferior reaction to the right on the tipping table and it is possible that this spontaneous sign influences the reaction. The induced deviation in the arms both on caloric and rotatory stimulation is not clear to either side with either arm and the pointing is sometimes crossed to both sides. The other cases display weak, vague reac-

Table 17.

Patient No.	Spontan. symptom	Diss. react. on caloric stimulation of (semicirc. canals)	Extent and direction of extremity reaction defect	Diss. react. on rotatory stimulation of (semicirc. canals)	Extent and direction of extremity reaction defect	Comments
33	Romberg: fall. tendency to r. Gait: deviat. to r.	horisontal (vertical not examined, r. labyrinth dead)	weak, indistinct, bilat., to both sides	vertical + horisont.	greatly reduced, indistinct, bilat., to both sides	Aged 65. Tipping react. stiff, spastic. Fall. tendency more pronounced to r.
57	0	vertical (+ horisont.)	greatly reduced, no distinct react., bilat., to both sides. Delayed, uncertain react. from horisont.	vertical	absent, bilat., to both sides	Aged 41. Encephalogram: cerebral atrophy, ext. hydrocephalus.
60	Romberg: fall. tendency backwards. Gait: no decid. deviat.	vertical	greatly reduced, indistinct, bilat., to both sides	»	absent, bilat., to both sides	Aged 52. sluggish, retarded, stiff tipping react.
64	Ataxia: r. arm. Point: lowers r. arm. Romberg: fall. tendency to l. Gait: no decid. deviat.	»	retarded, sluggish, reduced, crossed, bilat., to both sides	»	reduced, crossed to l. side, bilat., atactic movements	Aged 37. Tipping react.: stiff, sluggish, atactic, and slightly poorer to r.

tions in both arms and to both sides without crossing. The dissociated reactions are obtained in these four cases exclusively by stimulation of the vertical semicircular canals.

e. *Type IV.*

This type group comprises the cases which react with stiff, late and rigid extremity reactions. The patient stands almost like an inanimate object on the table and tips over if the movement is complete and rapid. It is not always of course that the reaction is so pronounced for there is no distinct borderline between this and the other types. See table 17.

No. 57 is a typical case of this group. The patient, aged 41, was

examined three years after the accident. The encephalogram shows atrophy of the brain, external hydrocephalus, and the patient exhibits no spontaneous signs. One obtains no extremity reactions to caloric and rotatory stimulation of the vertical semicircular canals, and late and uncertain reactions from the horisontal semicircular canals. Both mental and somatic reflexes and reactions are delayed and sluggish. No. 33 is a comparatively old man, sixty-five years, and it is possible that the reaction is due in part to senile changes.

It is not known why the patient reacts in this way to the tipping movements, but usually there is a general deterioration in the reflex releasing mechanisms, the reflexes being late and sluggish and approximately the same on both sides as in Nos. 33 and 57. A non-vestibular factor is thus introduced to explain this particular reaction. There is no particular spontaneous sign common to all of them. The dissociated reactions are usually released both from the vertical and horisontal semicircular canals, though preferably the former. The induced extremity reaction is usually delayed and the movements are stiff and rigid. The deviation is reduced or absent in both arms and to both sides. Apart from this stiffness and rigidity there is no special feature characteristic of this type and sometimes the examiner is surprised to find the patient reacting in this particular way.

In reply to the two questions put forward on page 105, one can only say that when one obtains dissociated vestibular reactions embracing pathological extremity reactions from the vertical semicircular canals only, or their central connections, one can expect a reaction as in types I, III and IV. In these types the defect is usually located only in the functions of the vertical semicircular canals. The induced deviation and pointing involve as a rule both arms more or less equally and the defect is equally pronounced to both sides. The spontaneous signs, particularly standing and the gait, show in these types no pronounced falling or deviating tendency to either side. Type IV is not so characteristic as I and III with regard to the vestibular reactions. The vertical semicircular canals always reacted the worst, but a nonvestibular factor must be reckoned with in the tipping reaction. The cases are too few for one to draw any conclusions from the examination.

With regard to type II, one finds here almost the reverse —

particularly to types I and III — in the matter of spontaneous signs. The induced vestibular reaction is more complicated but not so regular that one can draw any certain conclusions from the examination. Usually the reaction is defective to stimulation both of the vertical and horisontal semicircular canals, and the defective reaction in the arms is often directed to the side to which the tipping reaction is the best. As regards spontaneous signs, the walking test often displays a deviation to the same side as the falling tendency during tipping, and in some cases this applies also to Romberg's test. One obtains the impression that there exists a certain correlation between the direction of the decided falling tendency and the deviation shown in the Romberg and gait tests and the direction of the falling tendency on tipping. The series is too small for one to judge the matter definitely, but one can say that if one comes across clear spontaneous signs like those mentioned above then one can expect a tipping reaction as in type II, on condition that dissociated vestibular reactions with pathological extremity reactions can be demonstrated.

Type IV is characterized more by a reaction on the tipping table which is influenced by changes of a very different nature than those mentioned here in connection with the other types. The influential factor here is not of vestibular origin.

6. Regarding the Dominating Phases of the Tipping Reaction in the Different Types.

The various types of pathological tipping reactions have now been described. The difference between them is often not so large or so definite that one can immediately place a case in one of the types on the lines given here. One should therefore look for something more fundamental in the tipping reaction itself which can provide the basis for another classification but which need not for that reason displace the previous type grouping. For the object is not to divide the cases into these various types in practice. The vital thing is to find the pathological tipping reaction and one can then remember these various types as a help for one's own judgment. It is more important to see *how* the patient reacts in the first and final phases. Does the patient display better equilibrium reactions at the end or the beginning of the tipping reaction? What do the extremity reactions look like particularly with regard to coordination, symmetry, synergia, etc.? When is the extremity

Table 18.

Type of tipping reaction	Total	No. of cases		
		First phase dominating	Final phase dominating	Both phases equally dominating
I.	8	8	0	0
II.	25	1 patient No. 47	2 patient No. 7. 9.	22
III.	4	0	4	0
IV.	4	0	4	0

reaction released in relation to the table movement, and when is the patient's balance stabilised in relation to the stopping of the table movement? These are fundamental considerations in the pathological tipping reactions and if one considers these questions one obtains three groups or types of reactions. The first comprises defective reactions in the first phase, the second corresponding reactions in the last phase and the third a combined form. If one divides up the material on these lines one finds that there is no case with a defective first phase solely. The latter occurs only after purely peripheral vestibular destruction and there is no such case in the series.

Similarly there is no case in this series which reacts pathologically only in the final phase, for the first phase is always defective in one way or another, being frequently both retarded, insufficient and badly coordinated.

Both the first and final phases must therefore be regarded as pathological in all the cases and the examination is therefore designed to discover in which phase the defect is greatest and how it is expressed. In the foregoing table, the various cases in the different types have been grouped in this way in columns for "First phase dominating", "Final phase dominating" and "Both phases equally dominating".

In type I, one sees that the poor or delayed first phase is predominant and sets its stamp on the reaction, which includes a falling tendency at a comparatively early stage. In type II the situation is almost the reverse. The largest number of this type of cases are in the last column, showing no predominant phase in the

reaction. Two have a predominant final phase and one a predominant first phase. In the two last types all the cases are entered in the column for a predominant final phase, which is particularly marked in these cases. This applies especially to type III in which we find a particularly prominent cerebellar ataxia in the tipping reaction.

7. The Age of the Patient and of the Injury in Relation to the Phases of the Tipping Reaction.

When one observes this predominance of the different phases of the reaction one cannot but wonder what is the significance of the length of time between the accident and the examination. In cases examined at an early stage one would think that the defective first phase should be predominant. For no compensation can have occurred even if the vestibular injury is purely central, in analogy with a peripheral injury where healing or compensation passes through type I to type II and then to restitution in respect of the dissociated vestibular syndrome. Furthermore, the cases which react with a predominant final phase should perhaps for the same reason be older. An attempt will be made to clarify these matters in table 19.

As the table shows, the first phase defect is not only linked with older injuries; on the contrary it seems as though a predominant final phase is more usual in these cases. The distribution over different age groups is more even. The series is a small one, however, and one cannot draw any conclusions from these results but must attribute them to chance. The object of the table is merely to provide an answer to the first question above. With regard to the other question, the table shows that the assumption on which it was based is without foundation. Nor was anything else to be expected, and the series cannot provide any definite information in this respect because it is too small to allow of generalisations.

In order to see whether the age of the patient can affect the reaction, the series has been divided into age groups in this table. Most of the cases fall into the two central age groups. The phases are relatively evenly distributed in the different age groups.

Zador (102, p. 146) looks for variations in the tipping reaction between the two extremes of normal and wholly absent defensive movements and points out that if such could be discovered it

Table 19.

Approximate interval between injury and examination	Age of patients at examination					
	15—24 years	25—34 years	35—44 years	45—54 years	55—64 years	65—74 years
1 month						=
2 months				—		
4 »		—	—, —		—, +	
5 »			=		+	
6 »				=, —, —		
7 »			=			
10 »			=, =			
1 year			=, =		=	—
1½ years			=, =, +	=, =		
2 »	+, =	=, =	=			
3 »		=	+, —	—, =		
4½ »				=		
5 »			—			
6 »		+				
7½ »		=				
8 »				+	—	

Dominating first phase signified by . . . . . + (9 cases)
» final » » » . . . . . — (10 »)
Both phases equally dominating signified by . . . . . = (22 »)

would enlarge our knowledge of the tipping reactions. Post-traumatic vertigo yields just such reactions to tipping table examination. It is possible to study the individual features of the reactions, which are relatively constant, and in some cases it is possible to observe the healing of the patient's equilibrium defects and see the improvement in the tipping reaction at the same time as one observes the development of the dissociated vestibular reactions to a normal condition. They usually accompany each other, but one often obtains the impression that the tipping table examination is a somewhat more sensitive method.

Table 20.

Approximate interval between injury and examination	Age of patient					
	15—24 years	25—34 years	35—44 years	45—54 years	55—64 years	65—74 years
1 month		a.		b.	a + d.	c.
1½ months			a.			
2 »				a., c.	e.	
4 »		a + b + c.	c., b + c.		a + c., c., b., b.	
5 »			b + c.		a + c.	
6 »			b + c., a.	c., c., c.		
7 »			b + c.			
10 »			a + c + d., c.			
1 year			c., c.		c.	c.
1½ years			a + c., c., c.	c., c.	b.	
2 »	b + c., b., c.	a., a + c., b., b., c., b.	c.	b., e.		
2½ »			b.			
3 »		a + c.	a + b + c., c., b., a.	c., c.		
4½ »				a., c.		
5 »			c.			
6 »		a + c + d.				
7½ «		c.				
8 »				c.	c.	

Pathолog. nyst. response . . . . . signified by a.
Vestibular tonus difference . . . . . » » b.
Dissociated vestibular reactions . . . . . » » c.
Eye deviation . . . . . » » d.
Reaction of »vascular» origin . . . . . » » e.

8. Regarding the Occurrence of Pathological Otoneurological Reactions in Relation to the Age of the Patient and of the Injury.

In the foregoing table the cases in groups 1 and 2 have been divided up on the basis of the interval of time between the injury and the examination and of the age of the patient at the

time of the latter. It shows the distribution of the pathological reactions observed in otoneurological examination.

This table was chiefly compiled to supplement the previous one. The pathological tipping reaction is linked with dissociated vestibular reactions with pathological extremity reactions (c.). The distribution of the vestibulo-spinal changes coincides with that of the tipping reaction data in the previous table, but in this table are added the vestibulo-ocular injuries (pure cases) and the two with lesions of "vascular origin" but without anything pathological in the rest of the otoneurological condition. The two central age groups contain the largest number of patients. The vestibulo-ocular and vestibulo-spinal injuries are intermingled regardless of the patient's age or the time which has elapsed between the accident and the examination. It is possible of course that a larger series would yield different results.

F. General Remarks about the Tipping Reaction.

The fundamental value of the tipping table examination in the case of a patient who reacts pathologically lies in the changes one can observe in the two phases of the reaction. It is not of particular importance whether the patient falls or not; the main thing is to discover *how* he reacts to the table movements. Are the defensive movements retarded or inadequate to one or both sides and are there atactic extremity reactions? In practice it is the study of the patient's reaction to tipping table examination on these lines which is of the greatest clinical value, for one then obtains some idea of the locality of the injury. The more pronounced are the defects in the first phase, the more the injury is a pure vestibular one affecting the inducement of extremity reactions. The more pronounced it is in the final phase, the more it is a cerebellar injury. There is probably always a cerebellar factor in a pathological tipping reaction, but it is not so often that it is predominant.

It is practically speaking only tipping to the sides through the patient's longitudinal axis (fronto-occipital) which yields valuable results. In this series I have only attributed value to this form of tipping and the results previously described are obtained by tipping through a longitudinal axis. One sometimes finds, however, in cases of vertigo linked with vertical nystagmus that one

obtains the same vertigo by transversal tipping in the direction which might release the corresponding nystagmus. The patient then says that he recognises his spontaneous vertigo when tipped in this direction.

With regard to the course and prognosis of the various vestibular symptoms in groups 1 and 2, nothing can be deduced for certain from this examination since the series is not sufficiently large. A number of cases have been examined again one month, one year or two years after the first examination and in most cases the condition was unchanged, but twelve cases were fully restored, showing a completely normal condition. Since it is not possible to observe every case from the beginning one cannot say anything for certain about the prognosis. In cases 7, 14, 19, 20, 25, 27, 34, 56 with the dissociated vestibular syndrome, later examination — in the fifth case three months and in the others six months or a year after the first examination — showed perfectly normal reactions. The dissociated reaction did not exist and the tipping reaction was also normal. The patient had no symptoms. Continuous examination at certain definite intervals might perhaps have revealed a correspondence in the reactions between the dissociated reactions and the tipping reaction during healing. The patients named (particularly no. 19) could be followed up because they were resident in Stockholm.

G. Reply to the "Hypothetic Questions".

On page 24 where the object of this work was mentioned, three questions were posed the replies to which really contain the fundamental features of tipping-table tests as a rational method of examining vestibular function. Its clinical usefulness in cases of vertigo after head injury is subject to these questions receiving a satisfactory answer.

The first is: *When is the tipping reaction pathological?* — Reply: it is pathological in cases where there is an organic change which damages the vestibulo-spinal tracts and pathways for the extremity reactions. This applies to central vestibular lesions, but the tipping reaction is also pathological in cases of unilateral peripheral vestibular injury as long as compensation does not occur from the other side. In cases of bilateral peripheral and vestibular destruction it is permanently pathological. In the vestibular sta-

tus all this is seen in the fact that deviation and pointing are not obtained or are otherwise pathologically changed when caloric and rotatory stimulation are applied.

The second question is: *Why is the tipping reaction pathological?* — Reply: the extremity reactions which are induced by tipping for the preservation of balance are defective owing to injury to the vestibulo-spinal tracts or pathways or peripherally in the vestibular area. The mechanism for releasing vestibular extremity reactions is injured at the same time as the cerebellar function for the coordination, synergia, symmetry, etc. of the induced extremity reactions into well-balanced functional equilibrium reactions is upset in one way or another. The tipping reaction is thus pathological because these vestibulo-cerebellar equilibrium reactions are pathologically modified.

The last question is: *Is there any correspondence between the reaction obtained by examination on a tipping table and that obtained by the classical vestibular tests?* — Reply: Yes. There is a correspondence between a pathological tipping reaction and a dissociated vestibular reaction which embraces pathological extremity reactions to caloric and rotatory stimulation and sometimes also to subcortical optomotor stimulation. It is constant, and these two pathological reactions which accompany one another constitute a joint set of symptoms which it is proposed to call the *"dissociated vestibular syndrome"*.

This must not be confused with BARRÉ's "dysharmonie vestibulaire" which need *not* be linked with any *induced* dissociated vestibulo-spinal reaction defects.

The correspondence between the pathological tipping reaction and the dissociated vestibular reactions embracing the extremity reactions is not complete for in a couple of cases we have found fully normal reactions to caloric stimulation on the tipping table but a total absence of all post-rotatory reactions. The patients suffered from cerebrospinal syphilis and the discrepancy was attributed to this fact. Similar differences in the reactions to caloric and rotatory stimulation have previously been found by, among others, KLEESTADT (46), RAUSCH (75), RUTTIN (77, 78) and KOCH (55) in cases of syphilitic infection but also in ones of multiple sclerosis. We have never seen it in cases with pure, uncomplicated head injuries.

Summary.

The present work is primarily designed to demonstrate the diagnostic value of the tipping reaction in the examination of cases of vertigo after head injury. It is also designed to clarify the relationship in these cases between the tipping reaction and other vestibular reactions demonstrated during otoneurological examination.

The first chapter comprises a critical analysis of earlier works about the tipping reaction.

The second chapter contains first a working hypothesis, in which three questions with reference to the tipping reaction are posed. This is followed by a survey of the series, the method of examination and the result of its application to the series. In addition, the tipping reaction is dealt with in relation to the various other observations made during otoneurological examination.

A series of 140 head injury cases of ages varying between 19 and 67 years has been examined. Sixty of the cases displayed an organic central vestibular disorder. In two cases there were signs of an intracranial disorder but no pathological vestibular reaction, and finally there were 7 cases which showed psychogenic tipping reactions, but a normal neurological status. The remaining 71 cases reacted perfectly normally.

All the cases were referred to me by a neurologist for examination and the somatic neurological condition was almost always normal.

The vestibular examination comprised both tests for spontaneous symptoms (p. 33) — tests for ataxia in the arms, pointing, Romberg sign, gait and spontaneous nystagmus — and for induced vestibular symptoms and reactions (p. 35) — optomotor, caloric and rotatory stimulation and tipping table examination (p. 39) — as well as examination for nystagmus and vertigo in different positions. In addition, hearing was tested (p. 92).

In the caloric and rotatory tests, weak stimulation was used as far as possible in order to bring out the details of the reactions. The method employed does not differ from the customary one.

The tipping table examination was carried out on a special table, for the construction of which a number of experiments were conducted on animals (p. 45). The method and construction correspond on the whole with that used by RADEMAKER and GARCIN. It is designed to obtain as pure rotation and little passive displacement of the patient as possible, thus emphasising the first and final phases in the reaction. It is only tipping through the patient's longitudinal axis which yields results of value, and the patient must be placed on hands and knees. By tipping is meant herein a rapid movement of the table, usually taking 6—7/16 seconds or less. A table movement slower than 8/16 seconds does not necessarily demand vestibular reactions for the release of the requisite defence reaction.

A tabular survey (p. 74) shows the results obtained in each case by this method and one can there compare the spontaneous and induced signs and reactions directly with the tipping reaction. Comparison shows that:

1. there is no appreciable correlation between ataxia in the arms and the manner of tipping reaction;

2. the same applies with regard to spontaneous past-pointing and an abnormal Romberg reaction. In some cases, however, one can discern a certain correspondence between the direction of the spontaneous gait deviation and the falling tendency during examination on the tipping table;

3. there is a direct correspondence between a dissociated vestibular reaction to caloric and rotatory stimulation, embracing a pathological extremity reaction, and a pathological tipping reaction. In some cases the same correspondence is obtained with subcortical optomotor stimulation.

This correspondence between dissociated vestibular reactions embracing a pathological extremity reaction and a pathological tipping reaction constitutes a set of symptoms which it is suggested should be named the *"dissociated vestibular syndrome"* (p. 96). The correspondence was constant and in several cases it has been possible during healing to observe the simultaneous return to normal of these two components.

Forty-one cases reacted pathologically on the tipping table. They were divided into four types (p. 104):

I. Falling tendency to both sides = 8 cases
II. » » » one side = 25 cases
III. Hypermetric or similar reactions = 4 cases
IV. Stiff, rigid or similar reactions = 4 cases

The "psychogenic" reaction has been included as a fifth form of reaction. It is typical and, together with the neurological, psychological and other vestibular signs, enables the examiner to expose simulation and exaggeration.

Cases of vestibulo-ocular pathological signs embracing a pathological nystagmus response (8 cases) and a vestibular tonus difference (11 cases) display a normal tipping reaction. If pathological vestibulo-spinal signs are found at the same time as the above then the tipping reaction is pathological (14 cases). Pure vestibulo-spinal injury was found in 27 cases — all with a pathological tipping reaction. There is a correspondence between the pathological vestibulo-spinal signs and the pathological tipping reaction.

In types I, III and IV the vestibular pathological reaction was almost exclusively linked with a defective function in the central pathways from the vertical semicircular canals on both sides. The extremity reaction defect in these cases comprised absent, strongly reduced, crossed or similarly changed deviation in both arms and to both sides after both caloric and rotatory stimulation. In type II the corresponding reaction was as a rule linked with the same defect of function from both the horisontal and vertical semicircular canals. The reaction defect in the arms was approximately the same. Direct correspondence between this defect and the falling tendency on the tipping table was not observed for certain; the latter was not, however, very prominent. No direct explanation can be given of why the patient reacts less well to one side on the table. There is a certain degree of correspondence here with spontaneous walking deviation.

All that one can say is: If the patient reacts pathologically on the table in accordance with type I, III or IV, then one can expect a pathological reaction from the connections of the vertical semicircular canals for the inducement of extremity reactions to caloric and rotatory stimulation. If he reacts as type II one can expect the corresponding reaction to stimulation of all the semicircular canals.

The main thing, however, is the observation of the reaction in the first and final phases. Both are modified as a rule, but one

of them often dominates. The more the injury is of vestibular origin, the more the defect in the first phase becomes apparent, and the more it is of cerebello-vestibular origin the more hypermetria, assynergia and in-coordination set their stamp on the final phase. For diagnostic purposes, this observation yields the most, for it can expose a cerebellar injury which cannot otherwise be found. It is made manifest by the vestibular stimulation and takes the form of a defect in adjustment and adaptation to the new position created by the tipping. The vestibularly induced extremity reactions cannot be coordinated by the cerebellum into well balanced equilibrium reactions. This can be beautifully shown with moving pictures. — — — Type I embraces only cases with a predominant first phase and types III and IV only a predominant final phase. In type II the phases were equally prominent in 22 cases.

The predominance of one phase is not linked with the age of the injury or of the patient (p. 119). — — — The same applies to the pathological otoneurological reactions (p. 121).

The work concludes with a reply to the "hypothetic" questions (p. 123):

1. *The tipping reaction is pathological in the cases where* there is an organic change which injures the vestibulo-spinal tracts and pathways for the inducement of extremity reactions necessary for the preservation of balance during rapid tipping. This applies to central vestibular lesions. Where there is a unilateral, peripheral, vestibular loss of function the tipping reaction is pathological as long as no compensation has developed. In cases of bilateral peripheral loss of the vestibular function the reaction is permanently pathological.

2. *The tipping reaction is pathological because* the extremity reactions which are induced by the table movements are defective both in that they are too late or insufficient and that they are not functionally coordinated and synergised into efficient defence reactions. A vestibular and a cerebellar factor build up the normal tipping reaction in close cooperation.

3. *There is a correspondence* between on the one hand a pathological tipping reaction and on the other a dissociated vestibular reaction which embraces pathological extremity reactions to caloric, rotatory and sometimes subcortical optomotor stimulation. Exceptions to this were seen in two cases of cerebrospinal syphilis. The discrepancy was assumed to be due to this disease.

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